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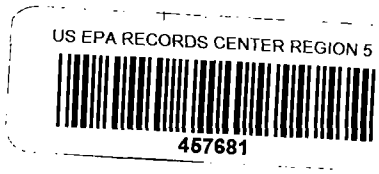

PITT-05-13-058

May 30, 2013

Project 112G01489

Department of the Navy
Naval Station Great Lakes
NAVFAC MW Code EV
Attn: Terese Van Donsel
201 Decatur Ave.
Building 1A
Great Lakes, IL 60088

Reference: CLEAN Contract No. N62467-04-D-0055
Contract Task Order 510

Subject: Final Remedial Investigation/Risk Assessment Report
Sites 9 – Camp Moffett Ravine Fill Area
Naval Station Great Lakes
Great Lakes, Illinois

Dear Mrs. Van Donsel:

Please find attached two hard copies and two compact discs (CDs) of the subject report for your files. Copies have also been distributed to the other members of the Naval Station Great Lakes Team as indicated below.

Also attached is one copy of the responses to Illinois Environmental Protection Agency comments. If you have any questions regarding the report, please contact me at 412-921-7251.

Sincerely,

Robert F. Davis, P.E.
Project Manager

RFD/alk

Enclosure

cc: H. Hickey, Naval Station Great Lakes/NAVFAC Midwest (1 copy with 1 CD and 1 RTC)
B. Conrath, Illinois EPA (3 hard copies with 3 CDs and 1 RTC)
O. Thompson, EPA Region 5 (2 copies with 2 CDs and 1 RTC)
J. Trepanowski, Tetra Tech (letter only)
File 112G01489/CTO 510, Tetra Tech (1 copy with 1 CD and 1 RTC)
G. Wagner, Tetra Tech – Administrative Record (1 unbound copy with 1 CD)

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RESPONSES TO ILLINOIS EPA COMMENTS RECEIVED MARCH 29, 2011
DRAFT REMEDIAL INVESTIGATION REPORT FOR THE
SITE 9 – CAMP MOFFETT RAVINE FILL AREA
NAVAL STATION GREAT LAKES
GREAT LAKES, ILLINOIS

Specific Comments

1. **Comment:** Executive Summary – Beginning in Section E.5, it appears all of the collected data reported here, for both soil and groundwater, have been compared only to Illinois EPA's Tiered Approach to Corrective Action Objectives regulations. According to the approved Sampling and Analysis Plan (SAP), the Project Action Limits were defined as "the more stringent of the USEPA Regional Screening Levels for Chemical Contaminants at Superfund Sites or Illinois risk-based criteria (Tiered Approach to Corrective Action Objectives [TACO])." The Executive Summary should also provide discussion comparing the data to the project action limits as defined in the SAP.

Response: Soil and groundwater results were compared to the criteria provided in Tables 4-3 and 4-6, as summarized in Section 4.0. It is correct that Executive Summary only provides a summary of the soil and groundwater results compared to TACO criteria. Text will be added to the Executive Summary to say the laboratory results were compared to USEPA and Illinois EPA TACO screening criteria. The following text will be added "Soil and groundwater analytical results were compared to risk-based screening criteria from both regulatory and non-regulatory sources to facilitate prompt identification of contaminants and exposure areas of concern that warrant further review and evaluation as part of the baseline risk assessment. Analytical concentrations were further compared to federal and state regulatory criteria, including the Illinois EPA Tiered Approach to Correction Action Objectives (TACO) criteria for ingestion and inhalation exposure, to highlight those locations at the property where concentrations exceed levels that are generally considered unacceptable for unrestricted use. The results of the comparisons to the TACO Ingestion and Inhalation Remediation Objectives for residential and commercial/industrial receptors for soil and groundwater are summarized below and in detail in Section 4.0."

E.6.1 Soil

The initial comparison of the subsurface soil results to the minimum risk-based screening criteria from Illinois EPA or the USEPA identified many exceedances. The minimum screening criterion in many cases is the soil to groundwater criteria provided by Illinois EPA TACO or USEPA (Section 4.3). However, when the soil results are compared to the TACO Residential and Industrial Ingestion and Inhalation screening criteria (Section 4.5), there are only a handful of exceedances as summaried below. Remediation objectives for the soil to groundwater pathway were only considered when contaminants were identified in groundwater at levels in excess of regulatory criteria.

2. **Comment:** Executive Summary – The second paragraph of Section E.1 concludes by stating that no ecological evaluation is necessary. It would be more satisfactory to verify that any contaminant contribution from Site 9 shallow groundwater to Pettibone Creek will be assessed during evaluation of the adjacent site which contains the headwaters of the creek.

Response: The following text will be added to the Executive Summary: "While the industrial nature of the site and limited habitat rule out any significant on-site ecological risk, the site investigation also considered whether the movement of site groundwater could pose an ecological threat to off-site areas. Groundwater from the site may be collected in the stormwater piping that discharges into the unnamed tributary to Pettibone Creek east of Sheridan Road. If the groundwater is contaminated it could contribute to surface water contamination in the tributary. However, investigations of Site 17, Pettibone Creek, and the unnamed tributary of Pettibone Creek did not identify surface water contamination or identify the Site 9 groundwater as a potential

contamination source. Site 17 included an ecological risk assessment and no chemicals detected in the surface water were retained as ecological chemicals of concern. A few chemicals were included in the food chain modeling; however the drinking portion of the model was an insignificant component of exposure because of the low detections in surface water compared to the sediment".

3. **Comment:** Executive Summary – The last sentence in Section E.6 discussing Soil, mentions the deposition of fill material into the Site 9 ravines "after placement of the subsurface piping". Explain the location and purpose of the subsurface piping.

   Response: The subsurface piping was placed in the bottom of the ravines in 1942 based on historical drawings (February 1942 Storm & Sanitary Sewer & Water Distribution drawing) in the area of Site 9 Camp Moffett and is listed as storm sewer pipe. The subsurface piping (identified as a 36 inch diameter reinforced concrete pipe for the northern and southern ravine and connects to an existing 54 inch diameter storm sewer on the drawing), was probably placed in the ravine to convey the unnamed Pettibone Creek tributary and to collect storm water from the Camp Moffett area after the ravines were filled in. Based on a 1945 drawing, the drawing indicated the ravine was to be filled in. The Initial Assessment Study indicated "examination of older aerial photographs and topographic maps of the area suggests that the area was formerly a narrow, V-shaped ravine, a former tributary of Pettibone Creek" confirming the subsurface piping was placed in the ravine to convey the unnamed Pettibone Creek tributary (see Appendix A of the report). No change will be made based on this comment. Also see the response to Comment 32 below.

4. **Comment:** Section 1.1 Project Overview – The first sentence should read "...to determine the nature *and extent* of fill materials ..."

   Response: The text was revised based on the above comment.

5. **Comment:** Section 2.3 – In the table on page 2-6, the last entry under Activity states that approximately 45% of the middle finger of the ravine is located under buildings at Site 9. This statement is inaccurate. Please review and revise as necessary.

   Response: The text was revised to say "Buildings at Site 9 are located over approximately 45 percent of the three filled in ravines."

6. **Comment:** Section 4.1 – The geophysical survey is discussed at the end of this section. However it is unclear what the result of that investigation was. Did the geophysical survey and the soil sampling determine the geographical boundary of the ravines? Was that goal accomplished? This needs to be addressed.

   Response: The Executive Summary (E.4), Section 3.3.3, and Section 4.1 were reviewed and revised based on this comment. Based on the geophysical survey results soil boring locations were placed in areas thought to be both inside and outside of the former ravines, in locations with potentially buried metallic objects, zones of small GPR reflectors, and areas of buried metal. There were correlations observed between the geophysical data and the location and alignment of the storm sewer in the northern ravine and the geophysical data and soil compositional (lithological) differences for the areas containing fill (gravel, sand, fly ash, coal, brick fragments, cinder, glass) in the three former ravines. Differentiation between fill areas was evident from an analysis of the geophysical data and soil borings information especially at the soil borings where the three ravines meet. Modern cultural interferences (such as buildings and aboveground metal objects) and reworking in the subsurface for the area may have contributed to masking former ravine areas and fill areas in the geophysical data.

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7. **Comment:** Section 4.4.4 – This section reports that dioxin/furan contaminants were not detected at concentrations exceeding the minimum regulatory screening criteria. However, the Draft Site Inspection Report, which used the exact same data set, reported an exceedance. Please explain why that exceedance is not reported here.

   Response: The calculation of the dioxin/furan toxic equivalent (TEQ) exceeded screening criteria solely because the calculation assumed the detection level for the non-detects. However, the TEQ is calculated for HHRA purposes and should not have been included in the Draft Site Investigation Report Section 5. No change will be made based on this comment.

8. **Comment:** Sections 4.5 – This section compares site soil and groundwater concentrations to TACO objectives. The title specifies two receptors, residential and industrial. Since the construction worker receptor is included in the risk assessment, please explain why the TACO construction worker objectives are excluded from this comparison.

   Response: The TACO and non-TACO Ingestion and Inhalation Soil Remediation Objectives for the Construction Worker will be added as a screening value to Tables 4-3 and 4-4. Section 4.5 will become the Soil Results Comparison to Illinois EPA Ingestion and Inhalation Remediation Objectives for Soil and Section 4.6 will become the Groundwater Results Comparison to Illinios EPA Ingestion and Inhalation Remediation Objectives. Text and tables has been added to Section 4.5 and 4.6 related to the comparison to the construction worker criteria.

9. **Comment:** Sections 4.5.1 – The last line in the paragraph concerning dibenzo(a,h)anthracene misstates the TACO objective. It should be reported as 90 µg/kg.

   Response: The text was revised based on the above comment.

10. **Comment:** Table 4-3 – Beginning with this table and continuing through all tables that present screening values, the application of a safety factor of 10 to the individual screening values needs to be standardized and documented, preferably on the table. As presented, there is uncertainty whether the one-tenth rule has been applied. It would be helpful as well if the trigger for applying the one-tenth multiplier were explained.

    Response: The safety factor of 10 is used as part of the risk assessment process that is described and used in Section 6 of this report. USEPA RAGS does not explicitly state the safety factor of 10; however, in the link to a USEPA memo on the RBC table it discusses the safety factor of 10 (http://www.epa.gov/reg3hscd/risk/human/info/cover.htm - see the FEATURES AND HISTORIAL CHANGES section). The web page explains how you should divide by 10 to adjust the criteria for an HQ of 0.1. The text from this section is below:

    "At Region III Superfund sites, noncancer RBCs are typically adjusted downward to correspond to a target HQ of 0.1 rather than 1. (This is done to ensure that chemicals with additive effects are not prematurely eliminated during screening. Note that the RBCs displayed on the table are shown at an HQ of 1; to arrive at the RBC at 0.1, data users must do the conversion themselves.) However, some chemicals have RBCs at HQs of 0.1 that are lower than their RBCs at 1E-6 cancer risk. In other words, the screening RBC would change from carcinogenic to noncarcinogenic. These chemicals are flagged with a "!" symbol. Therefore, assessors screening with adjusted RBCs will be alerted to this situation. See the companion attachment to the RBC Table, "Alternate RBCs," for alternate values for "!" RBCs."

    The safety factor of 10 does not apply to any tables in Section 4.0. No change will be made based on this comment.

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11. **Comment:** Tables 4-6 and 4-7 – Table 4-6 lists the screening criteria for groundwater and Table 4-7 presents the occurrence and concentration summary also for groundwater. Earlier, Tables 4-4 and 4-5 presented corresponding information for soil. Tables 4-4 and 4-5 include the same chemical parameters yet the lists of contaminants differ between Tables 4-6 and 4-7. Please explain or correct this discrepancy.

    Response: Table 4-6 was corrected based on the above comment.

12. **Comment:** Section 6.4.2.3 – The paragraph addressing Navy recruits as possible receptors mentions that Site 9 has been covered with clean soil. This is the type of information that we are asking to be summarized in the General Comment below to support excluding surface soil from this evaluation.

    Response: Comment noted. See the response to the General Comment 33 below.

13. **Comment:** Section 6.4.5.1 – The averaging time (AT) factor in the dermal contact with soil equation incorrectly indicates that the non-cancer AT should be converted to hours.

    Response: The text has been revised based on the above comment. AT is converted to days.

14. **Comment:** Section 6.7.2.2 – This section discusses the bias due to sampling in the ravine where waste may have been placed and reasons that such a practice likely overestimates the risks. That may be true, but the lack of sample locations due to inaccessibility (location of buildings and locations off-site) could well have the reverse effect and underestimate the risks. This should be stated as well.

    Response: The text has been revised based on the above comment. The following text was added "In addition, lack of sample locations due to inaccessibility (location of buildings and locations off-site) may lead to under or over estimate of the risk to potential receptors."

15. **Comment:** Section 6.7.2.3 – The last paragraph of this section makes a comparison of the average inorganic compound values to the background values. Arithmetic means are generally unacceptable for use as exposure point concentrations in human health risk evaluations. The procedures outlined in the USEPA ProUCL user's guide should be followed.

    Response: Arithmetic means were not used as exposure point concentrations. Appropriate and applicable USEPA ProUCL guidances were followed in the risk assessment analysis. As stated in this section, "No chemicals in soil and groundwater were eliminated as COPCs on the basis of comparisons to background concentrations." It is understood from the Illinois EPA TACO background criteria in Appendix A, Tables G and H is the upper limit of the area background concentration for the site. However the comparison to both maximums and averages of the inorganic COPCs to background criteria was simply included as a point of discussion in the Uncertainty Section of the risk assessment to indicate how the inorganic compounds may affect the risk estimates and the conclusions of the risk analysis. No change will be made based on this comment.

16. **Comment:** Table 6-2 – This table is a good example of the inconsistency observed in applying the one-tenth rule; TACO values are not factored, USEPA values are factored, and vapor intrusion values are not factored. Use of the one-tenth factor appears to be arbitrary.

Response: See the response to Comment 10. There is no inconsistency in applying the one-tenth rule. As was explained in previous Naval Station Great Lakes risk assessments and their work plans (Site 19, Site 5, etc.) only risk-based non-carcinogenic criteria are divided by ten to correspond to a systemic hazard quotient of 0.1 for risk-based screening levels of non-carcinogens. Therefore, each set of criteria and each individual chemical within each set of criteria, are assessed individually to determine if it is appropriate to be divided by 10. For example, Illinois EPA TACO soil non-carcinogenic criteria and USEPA soil non-carcinogenic criteria (residential, commercial, industrial, construction worker) are divided by 10 because they are risk-based values. However, TACO soil to groundwater migration screening values are not divided by 10 because these are based on acceptable target groundwater concentrations that are primarily groundwater standards, not straight risk-based concentrations. In addition, TACO Objectives for Groundwater are based on the Illinois State Groundwater Quality Standards, which reference the federal MCLs (drinking water standards), therefore are not divided by 10. USEPA-ORNL tap water values are straight risk-based values, so non-carcinogenic criteria are divided by 10. Again, each chemical and set of criteria is individually evaluated and only risk-based non-carcinogenic criteria are divided by 10 to correspond to a hazard quotient of 0.1. No change will be made based on this comment.

17. **Comment:** Table 6-5 – This table presents the exposure point concentrations (EPC) to be used in the risk calculations. Please explain how both total TCDD equivalent concentrations can be lower than the single 2,3,7,8-TCDD value. Also, explain the absence of a 2,3,7,8-TCDD EPC for the central tendency exposure (CTE) receptor.

Response: The source of this apparent discrepancy is that the 2,3,7,8-TCDD congener was detected in only 2 of 7 samples, while the 7 samples had detections of some dioxin/furan congeners. Because there were only 2 detected results for 2,3,7,8-TCDD, meaningful statistics (including mean and 95% UCL) could not be calculated; therefore, the maximum detected concentration was listed as the RME Exposure Point Concentration (EPC) in Table 6-5. The RME EPCs for total 2,3,7,8-TCDD TEQs represent the calculated 95% UCL of TCDD TEQs for the 7 samples, and these are lower than the maximum 2,3,7,8-TCDD analytical result. No change will be made based on this comment.

18. **Comment:** Table 6-8 – The full citation for footnote #3 should be added to the reference section of the report. The exposure duration (ED) for the occupational/maintenance worker receptor is reported here as nine years. USEPA's guidance document "Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposures" (1993) suggests a default ED of five years and averaging time of 1825 days for this receptor. Please explain the differences between the report and the guidance document.

Please explain the four hour exposure time (ET) for dust and volatiles from soil by the CTE occupational/maintenance and construction workers. Inhalation of fugitive dusts and volatiles from soil is a passive exposure. These exposures are controlled by meteorological conditions, physical properties of the chemicals and soil, and soil contaminant concentrations. The only reasonable justification for the ET to be halved is if the central tendency receptor spends one-half day at the site. If this is the underlying assumption, it should be stated and justified.

Response: The full citation for footnote #3 has been added to the Reference section.

It should be noted that the CTE occupational ED of 9 years was presented in the Work Plan. However, the 1993 guidance document was a draft document and has been superseded by later guidance, such as USEPA Exposure Factors Handbook (1997). Table 15-176 of the USEPA Exposure Factors Handbook (1997) provides a CTE duration for occupational receptors of 6.6 years. The 9 year exposure duration that was used in the risk assessment for the occupational

receptors is somewhat more conservative than the more recent guidance. This will be discussed in the uncertainty section, and no change is proposed to the risk assessment calculations for Site 9. Future risk assessments will use the USEPA 1997 CTE exposure duration of 7 years (i.e., 6.6 years from Table 15-176 rounded to 7) for occupational receptors.

The Risk Assessment Work Plan indicated that professional judgment would be applied for some CTE values for which there are no defaults. The justification for attributing one-half day for the inhalation pathway is that it is reasonable for typical occupational workers at the Navy base to spend less than all day outside, even if they perform maintenance duties. No change will be made based on this comment.

19. **Comment:** Table 6-9 – Numerous errors were noted on this table of non-cancer toxicity values for the oral and dermal routes of exposure. It is incumbent upon the Tier 3 applicant to provide the most current toxicity values available.

- Manganese: Change chronic oral reference dose (RfD) to 0.02 mg/kg-d. The Integrated Risk Information System (IRIS) documentation states that up to 5 mg/day of manganese is obtained from the diet; thus, half of the intake must be subtracted from the acceptable dose.
- Vanadium: Change chronic RfD to 0.00007 mg/kg. This is a Provisional Peer Reviewed Toxicity Value (PPRTV) available for elemental vanadium and vanadium compounds other than vanadium pentoxide. Documentation can be found at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php.
- Arsenic: We cannot verify the PPRTV subchronic RfD (RfDs) from October 2005. Alternative is HEAST 1997.
- Chromium VI: Change the RfDs to 0.005 mg/kg-day based on the Agency for Toxic Substances and Disease Registry (ATSDR) value.
- Naphthalene: Add RfDs of 0.6 mg/kg-d, ATSDR.
- TCDD: Add RfDs of 2.0E-08 mg/kg-d, ATSDR.
- Antimony: Add RfDs of 0.0004 mg/kg-d, PPRTV.
- Barium: Add RfDs of 0.2 mg/kg-d, ATSDR.
- Cadmium: Add RfDs of 0.0005 mg/kg-d, ATSDR.
- Cobalt: Add RfDs of 0.003 mg/kg-d, PPRTV.
- Copper: Add RfDs of 0.01, ATSDR.
- Iron: Add RfDs of 0.7 mg/kg-d, PPRTV.
- Manganese: Add RfDs of 0.02 mg/kg-d, chronic value.
- Selenium: Add RfDs of 0.005 mg/kg-d, HEAST.
- Vanadium: Add RfDs of 0.0007 mg/kg-d, PPRTV.
- Zinc: Add RfDs of 0.3 mg/kg-d, ATSDR.

Response: Many of Illinois EPA's chemical-specific toxicity criteria comments represent clarifications of the chemical-specific Tier 3 toxicity criteria sources (e.g., PPRTV and ATSDR) that Illinois EPA recommends. Furthermore, Illinois EPA's recommendations relate primarily to subchronic RfD values (for exposure durations that are 7 years or less), which apply only to the construction scenario. Please note that if a specific subchronic RfD was not listed in Table 6-9, the chronic RfD was applied in the risk assessment of subchronic exposure scenarios.

By and large, changing these toxicity values will have little impact on risk characterization summaries and certainly not on remedial decision-making. Therefore, the level of effort required to quantitatively revise the Site 9 Risk Assessment to address these comments is not warranted. Rather, we generally propose to address the differences in the Illinois EPA recommended toxicity

values qualitatively in the uncertainty section of the Site 9 risk assessment. The following section will be inserted into the Uncertainty Analysis section of Section 6 of the report.

"6.7.5.2 Impact on Risk Characterization if Illinois EPA Additional Toxicity Criteria Were Applied

During its review of the draft of this HHRA (Illinois EPA, 2011), Illinois EPA proposed a number of alternative toxicity criteria, primarily subchronic RfD from Tier 3 toxicity resources (per USEPA, 2003). Many of Illinois EPA's chemical-specific toxicity criteria comments represent clarifications of the chemical-specific Tier 3 toxicity criteria sources (e.g., PPRTV and ATSDR) that Illinois EPA recommends. Summarized below is a comparison between the toxicity criteria that have been applied in the risk assessment and those alternative toxicity values proposed by Illinois EPA. This analysis is presented in its entirety in Appendix G. As indicated by this evaluation, there would not be substantive changes in the overall risk characterization nor risk decision-making if the alternative Illinois EPA values are used.

Appendix G Uncertainty Table 1 presents the chronic and subchronic toxicity values proposed by Illinois EPA compared with these values that were used in the Site 9 risk assessment. For a number of COPCs, Illinois EPA's proposed subchronic oral reference doses, Tier 3 subchronic toxicity values, and RFCs/RFDs are higher than the values that were used in the Site 9 RA. These include chronic and subchronic RfD for TCDD, subchronic RfD for naphthalene and cobalt. Therefore the risks calculated for subchronic scenarios (i.e., the construction scenario) are more conservative in the Site 9 RA than if these subchronic values are applied for these COPCs. A number of the proposed Illinois EPA RfD values are the same as those used in the Site 9 risk assessment. These include the subchronic RfD for antimony, barium, iron, selenium, and zinc. There would be not change to the risk calculations in response to the comments on these chemicals. In a couple cases (for cadmium and copper), the proposed Illinois EPA subchronic RfD from a Tier 3 source was lower (that is more conservative) than the chronic RfD from Tier 1 source IRIS. Therefore, we propose that retaining the chronic RfD from IRIS as a surrogate for a subchronic RfD in these two cases. Finally, there were a number of Illinois EPA's proposed toxicity values that are more conservative than those used in the Site 9 RA. These include the chronic RfD for manganese and vanadium, and the subchronic RfD for aluminum, arsenic, and chromium VI.

In the Uncertainty Analysis in Appendix G, side-by-side comparisons were made between some original risk spreadsheets and spreadsheets modified with the Illinois EPA toxicity values. The scenarios evaluated are construction (i.e., the only scenario in which subchronic RfD values were applied) and the child residential scenario (a receptor who is more sensitive for evaluating noncancer exposures), Table 7.1 (Construction worker noncancer hazard from oral and dermal exposure to subsurface soil) shows the original HI calculated is 1.0. When all of the proposed Illinois EPA RfD values are applied, there is no change in the summed HI for this receptor. Likewise, original Table 7.3 (Construction worker noncancer hazard from dermal exposure to groundwater) and the revised spreadsheet with Illinois EPA RfD have the same summed HIs (0.1). For the hypothetical child residential receptor, Table 7.9 calculates noncancer hazards for exposure to subsurface soil through ingestion and dermal contact. The HI of 3.4 is similar but a little lower than the HI calculated for this receptor with the proposed Illinois EPA chronic RfD values (HI of 3.6). The original HI is greater than 1, so this slight increase in HI would not change the remedial decision-making based on this scenario. Likewise, the HI for the hypothetical child residential receptor from exposure to groundwater (HI of 12)

is slightly higher with the proposed Illinois EPA RfD values compared to the HI presented in the Site 9 Risk Assessment (HI of 11). However, the original HI is in great excess of 1, and the same remedial decision would be supported with modification of the RfD values.

In summary, the summed HIs for the construction worker and residential child are the same or very similar when the Illinois EPA toxicity values are applied compared to the summed HIs in the Site 9 RA. Therefore, changing these toxicity values would not result in substantive changes in the overall risk characterization nor risk decision-making, if the alternative Illinois EPA values are used."

Finally, a set of tables will be added to the end of Appendix G that supports the Uncertainty Analysis of use of different Tier 3 toxicity criteria. These are also included as Attachment 1 of this Response to Comments document. Attachment 1 includes G-1 that shows a comparison between the RfD values used in the original Site 9 risk assessment and those that Illinois EPA has recommended in its comments; the next eight tables provide side-by-side comparisons of original risk spreadsheets and risk spreadsheets with Illinois EPA-recommended toxicity criteria replacement. As noted, there is little difference in the summed HIs between the original risk calculations and those with Illinois EPA toxicity values, and the replacement of Tier 3 toxicity criteria makes no change to the remedial decisions for Site 9.

For future risk assessments, may we suggest the following procedure: After COPCs are identified for a risk assessment but prior to commencing with risk calculations, the Navy will provide Illinois EPA with the proposed toxicity criteria (chronic and subchronic oral RfD and inhalation RfC; oral cancer slope factors; and inhalation unit risks). We will request that Illinois EPA provide concurrence on these values, or recommend alternative values before further preparation of the risk assessment is made.

The following addresses the comments on specific toxicity values.

Manganese: This approach is not explicitly described in the IRIS for manganese; therefore, we do not propose to change the risk calculations of the Site 9 Risk Assessment based on this comment. However, we recognize that the User Guide to the USEPA Regional Screening Levels does prescribe subtracting the daily dietary contribution of manganese from the RfD (0.14 mg/kg-day/2 = 0.07 mg/kg-day), and then adjusting the new RfD for nondietary exposures by the modifying factor of 3 (0.07 mg/kg-day/3 = 0.024 mg/kg-day). Therefore, we agree to discuss this in the uncertainty section of the Site 9 report, and in future risk assessments to change the chronic oral RfD (and subchronic per "manganese RfDs" comment above) for manganese to 0.02 mg/kg-day.

Vanadium: It should be noted that one of the key differences between PPRTV values and IRIS values is the opportunity for public review and comment of draft IRIS values before they are finalized. No such unsolicited review is included for PPRTV values. Our review of the provisional toxicity criteria document referenced here shows that there is low confidence in the key study (Boscolo et al., 1994, which is also a subchronic study rather than chronic study) and therefore low confidence in the provisional subchronic and chronic RfDs. There is an uncertainty factor (UF) of 3000 applied to the No Observed Adverse Effects Level (NOAEL) to yield the very low provisional chronic RfD of 0.00007 mg/kg-day. The toxic effect (kidney cellular changes) was observed only in male rats, and this may be a gender/species-specific toxic effect and is common for male rats.

Recently the USEPA RSL guidance (May 2011) proposes the following approach to calculating a RfD for vanadium compounds other than pentoxide. According to the User Guide Section 5.4, "(t)he oral RfD toxicity value for Vanadium, used in this website, is derived from the IRIS oral RfD for Vanadium Pentoxide by factoring out the molecular weight (MW) of the oxide ion. Vanadium

Pentoxide (V205) has a molecular weight of 181.88. The two atoms of Vanadium contribute 56% of the MW. Vanadium Pentoxide's oral RfD of 9E-03 multiplied by 56% gives a Vanadium oral RfD of 5.04E-03." We propose no change in the risk calculations of Site 9, but to discuss in the uncertainty section the potential impact on the risk assessment conclusions if 0.005 mg/kg-day were applied as the oral RfD for vanadium. In future risk assessments, we propose to use the oral RfD of 0.005 mg/kg-day for vanadium.

Arsenic: The subchronic RfD is found in USEPA Region 8 (2002) Derivation of Acute and Subchronic RfD for Inorganic Arsenic. This paper has been included with this Response to Comments document. The reference in Table 6-9 will be changed to this Tier 3 source.

Various Subchronic RfD recommendations:
For the following COPCs Illinois EPA's proposed subchronic RfD are the same as the chronic RfD that were used in the Site 9 risk assessment: antimony, barium, iron, selenium, and zinc. Therefore there is no change in the risk assessment result for subchronic scenarios (i.e., the construction scenario) for these COPCs. For a number of other COPCs the proposed Tier 3 subchronic toxicity values are higher than those used in the Site 9 risk assessment (e.g., naphthalene, 2,3,7,8-TCDD, and cobalt), Therefore, the risks calculated for the construction worker would be more conservative in the Site 9 risk assessment, and this will be discussed in the uncertainty section. With regard to the Illinois EPA's comments on subchronic RfD for manganese and vanadium, please refer to the chronic RfD discussion above. The differences in professional judgment will be explained in the uncertainty section, as well. Finally, we disagree on the comment recommendations for subchronic RfD for cadmium and copper. In both of these cases the recommended subchronic RfD is lower (more conservative) than the chronic RfD provided in USEPA toxicity sources. Therefore, for cadmium and copper, we propose to maintain the use of the chronic RfD for the subchronic scenario.

20. **Comment:** Table 6-11 – The preferred oral cancer slope for TCDD is 1.3E+05 $(mg/kg\text{-}day)^{-1}$ from California EPA.

Response: It does not seem appropriate to use a California-specific toxicity value for a Site in another state when, as in this case, a USEPA toxicity value is available. Therefore, we propose to leave this unchanged in the risk assessment of Site 9. Please note that the suggested oral cancer slope factor is somewhat less conservative than the HEAST value that was used in the Site 9 risk assessment. No change will be made based on this comment.

21. **Comment:** Table 6-12 – The conversions of unit risk values to inhalation cancer slope factors are inappropriate for all but two chemicals (TCDD and TCE). Chemicals are not eligible for conversion when they induce tumors at the point of impact with the body. Furthermore, we observe that inhalation slope factors are not used in the Appendix G calculations of risk. Both the inhalation RfD conversion's column and the unit's column are unnecessary.

The inhalation unit risk value presented here for vanadium is actually for vanadium pentoxide. Vanadium metal and other vanadium compounds are not carcinogenic. The analytical results should be examined to determine which form of vanadium is present.

Response: The inhalation CSFs and their units columns have been removed from Table 6-12.

There is no process knowledge or historical waste disposal information to support that vanadium pentoxide (V_2O_5) is a predominant form of vanadium detected in soil samples. Therefore, we propose to discuss the conservatism of including vanadium in the inhalation risk calculations of the

Site 9 risk assessment. In future Great Lakes Naval Station risk assessments, toxicity values for vanadium and compounds other than vanadium pentoxide will be used.

22. **Comment:** Section 7.1 – This section is written much the same as the Executive Summary. As such, those same comments apply to this section as well.

Response: Revisions made to the Executive Summary will also be made to Section 7.0.

23. **Comment:** Section 7.1 – It states in the first paragraph that there was "some correlation between the geophysical data and observations from the soil boring investigation" for the three ravines. Please define "some correlation". Were the objectives for this portion of the investigation met? Were the geographical boundaries of the ravines accurately determined?

Response: See the response to Comment 6.

24. **Comment:** Section 7.2 – This section does not really present any conclusions based upon the risk assessment as is the title of this section. In fact, it does not even state that there are unacceptable risks at this site. There is only discussion of the site data when compared to background. There should be a fully developed discussion of what the results of the risk assessment mean, so that the following Recommendations Section can address how that risk may be eliminated or addressed, if necessary.

Response: The second and third paragraph of this section did summarize the COCs and the residential and construction worker receptors with the non-cancer (HQ>1) and cancer (>$1x10^{-6}$) risk in soil and in groundwater. Additional discussion of the risk assessment will be added to this section based on the comment, including a discussion of the results for the RME and CTE analysis, so that the recommendations can be developed. The following paragraphs will be added

Non-Carcinogenic Risks to Receptors

Adverse non-carcinogenic health effects are not anticipated for the occupational/maintenance workers in the study area since the risk assessment calculated RME and CTE HIs were less than or equal to 1.0. However the RME and CTE total HIs are greater than 1.0 for the future construction workers in the study area. If domestic use of groundwater is not considered a complete pathway, adverse non-carcinogenic health effects are also not anticipated for the future adult residents since the risk assessment calculated RME and CTE HIs were less than or equal to 1.0. However the RME HIs are greater than 1.0 for future child residents from the ingestion of subsurface soil but the CTE HIs for the future child resident are less than or equal to 1.0.

Carcinogenic Risks to Receptors

RME and CTE cancer risk estimates for construction workers and occupational/maintenance workers do not exceed the target USEPA cancer risk range (1x10-4 to 1x10-6). However, RME cancer risk estimates for construction workers and occupational/maintenance workers future exceed the Illinois EPA risk goal ($1x10^{-6}$). If domestic use of groundwater is not considered a complete pathway, the total site RME and CTE cancer risk estimates for total future residents (adult and child) are within the target USEPA cancer risk range ($1x10^{-4}$ to $1x10^{-6}$), but exceed the Illinois EPA risk goal ($1x10^{-6}$). The major contributors to cancer risk under this scenario are arsenic and PAHs in subsurface soil. However, it is probable that PAHs at the site are attributed to background.

25. **Comment:** Section 7.3 – Obviously, this section needs to be completed. The Agency would suggest waiting until the Remedial Investigation is complete and the risk assessment revised as necessary before developing any recommendations for this site.

    Response: Comment is noted. Based on the changes that were made to the RI/RA report for the Illinois EPA comments the following recommendation is added: "Based on the results of the RI it is recommended that a Focused Feasibility Study be performed for Site 9. Several remedial alternatives to mitigate risks will be identified and considered during preparation of the Focused Feasibility Study.

26. **Comment:** Appendix B-10 – The Chain of Custody forms provided here appear to be missing some information, are poorly copied, and are difficult to read. Suggest this section be reviewed and revised to correct these deficiencies.

    Response: Appendix B-10 will be reviewed and corrected as needed. Note that these are forms that developed in the field and corrections were made to the forms after they were printed to match the samples that were placed in the cooler.

27. **Comment:** Appendix G – The units are incorrect for the final intake result on all inhalation intake tables. The units should be "mg/m^3". This comment affects Tables 4.2, 4.2a, 4.5, 4.5a, 4.7, 4.10, and 4.10a.

    Response: The units have been corrected in the heading of the mg/m^3 columns of these tables.

28. **Comment:** Appendix G – Tables 4.7a, 7.7a, and 8.7a are missing from our copy of the report. They should be included to present the adult resident central tendency inhalation contact assumptions and calculations followed by tables of the intakes and calculated hazard quotients.

    Response: These tables will be provided in the final RI/RA report

29. **Comment:** Appendix G – Tables 4-11 and 4-11a present the intake calculations for the child receptor. For mutagens, age-related intake values are calculated. Typically, age-related intake variables such as water ingestion rate, surface area, and body weight are also selected to match the receptor's age. It appears that only the exposure durations have been adjusted in the subject tables.

    Response: The reviewer is correct for Table 4-11 that only exposure duration was adjusted. While for the 3-6 year old interval this yields a somewhat more conservative result, it is not discernable in the summed risk characterization. However, in future risk assessments, the intake calculations for the child receptor will be modified to incorporate the other age-specific exposure factors in addition to exposure duration. No change will be made based on this comment.

30. **Comment:** Appendix G – Tables 8-11 and 8-11a present risk calculations for the residential child receptor. We cannot establish that the age-dependent adjustment factors (ADAF) of 10X (ages 0-<2) and 3X (ages 2-<16) have been applied to the oral slope factors and unit risk factors when carcinogenic risks were calculated for the mutagenic contaminants. Please verify whether that was the case.

    Response: The age-appropriate ADAFs have been incorporated in the calculating risk spreadsheets (10x for 0-2 yr; 3x for 3-6 yr for the 0-6 year exposure duration of the child receptor)

of RME – Table 8-11. The cells containing these calculations were outside of the print area of the Table 4's, and are correctly incorporated into the Table 7's and 8's for the child receptor. In future risk assessments the ADAF cells will be labeled and included on these tables.

31. **Comment:** Appendix G – Footnotes "(1)" and "(2)" appear on all of the tables presenting the hazard and risk calculations in Appendix G. Please provide the denotations for these footnotes.

Response: (1) Specified Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation. (2) Subchronic values in italics. We propose to insert a sheet before the Appendix G tables that define these footnotes of these tables.

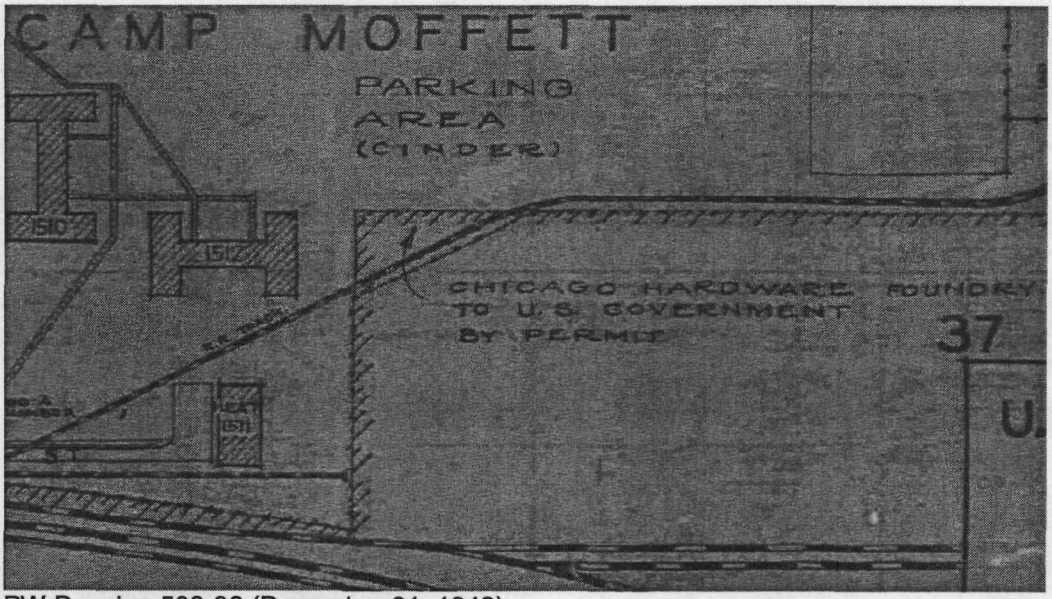
General Comments

32. **Comment:** Given that the RI reports in Section 7.1 that the "general area of contamination at the site based on the laboratory results appears to be where the three fingers of the ravine merge", and that all available evidence points to the fact that the ravine extends farther to the east at least as far as the roadway, the Agency believes additional investigation is required to verify the full extent of the ravine and to determine if there may be higher levels of contamination in the down-gradient direction within the ravine. Data needs include both subsurface soil and groundwater analyses. The Agency suggests at least four subsurface soil sample locations and two groundwater sample locations. Based upon the current figures, it appears that this additional investigation would be conducted entirely off-site on property not currently owned by the Navy.

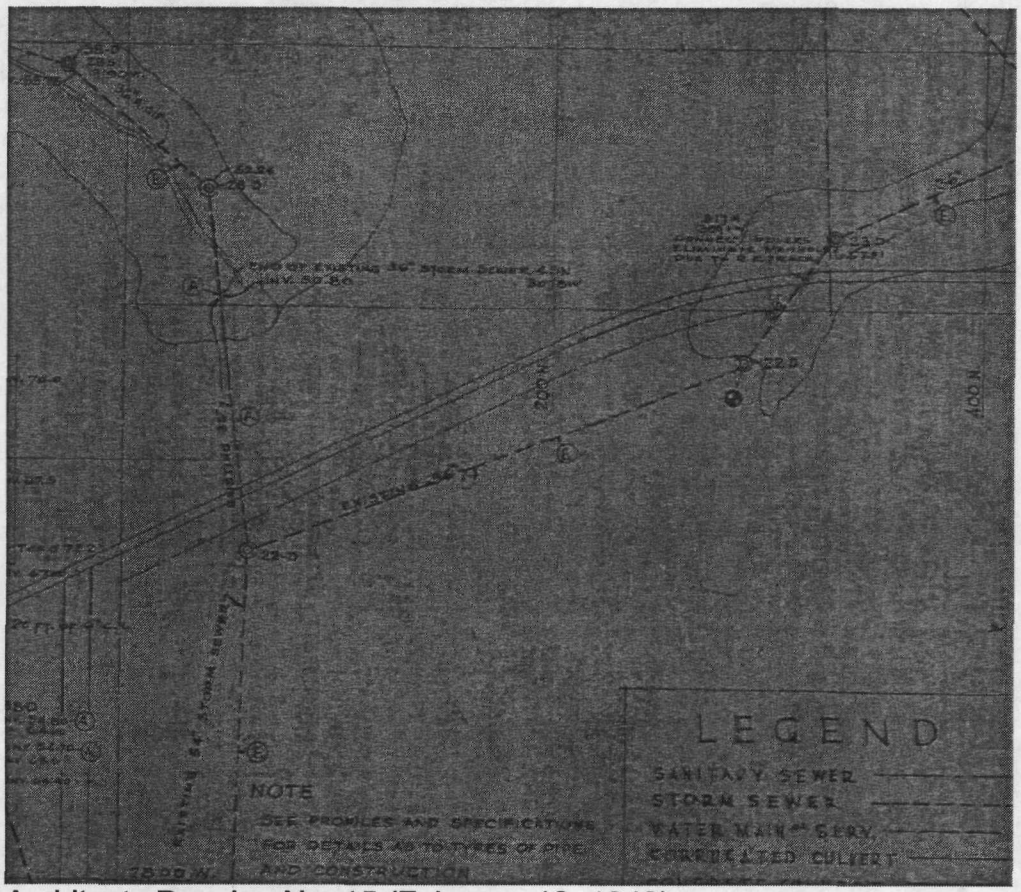
Response: Attached to this response to comments is a 1958 drawing showing the ownership information related to Naval Station Great Lakes. As shown on this drawing, the Site 9 Camp Moffett area was acquired by the Navy in 1918, transferred to the VA in 1924, occupied by Navy permit in 1942, and transferred back to the Navy in 1950. The area east of Site 9 Camp Moffett that is mentioned in this comment was not owned by the Navy; however the Navy did occupy a small triangular section of the property by permit starting in 1942 for a railroad spur that was used for the delivery of coal to the area of Site 21 (see part of PW Drawing 508-96 below). This neighboring property was owned by the Chicago Hardware Foundry Company. See portions of two different 1942 drawings below.

Based on other drawings (see the response to Comment 3) the ravine in the Site 9 Camp Moffett area was filled in after 1945, but the area was owned by the Chicago Hardware Foundary Company including the small triangular section of the property that the Navy received the permit to occupy in 1942 was filled in before December 1942 based on PW Drawing 508-96 which showed the conditions as of December 1942.

Even though it is known that the ravine extends farther to the east, Illinois EPA's request to conduct additional investigation of areas that are off-site of the Navy property and related to property that was not owned by the Navy, only occupied by the Navy for a railroad spur for the delivery of coal is not reasonable. This area was filled in before the Navy occupied a small portion of the property and it was owned by others.



PW Drawing 508-96 (December 31, 1942)



Architects Drawing No. 15 (February 12, 1942)

33. **Comment:** This report calculates human health risks for chemical contaminants in subsurface soils only, as was agreed upon during development of the Sampling and Analysis Plan. It is well established that surface soils are typically the major contributor to risk due to the high potential for direct contact. At Site 9, concern about surface soils now seems warranted since the single surface soil sample that was analyzed showed dioxins were present. A comprehensive discussion and evaluation of all information regarding surface conditions at Site 9 should be presented. In the absence of analytical results, comprehensive arguments and documentation supporting the safety of the surface soils should be compiled. However, since the results of this investigation dictate that additional sampling be conducted to determine the full nature and extent of contamination, a re-evaluation of this strategy may be necessary. A limited number of surface soil samples should be considered.

Response: The above comment is noted; however the history of the site is related to buried waste in the former ravines as was described in Section 1.1. Numerous construction activities and construction of new buildings and facilities have occurred at the site covering the fill materials with buildings, asphalt, concrete, and clean topsoil to landscape the area. The surface soil would not be representative of the buried waste and ravine conditions. The detected concentration of dioxin in the one surface sample is likely a background level considering this area was industrial with coal fired power plants and heating facilities, zinc smelters, and foundry type facilities. In addition, the dioxin concentration of the surface soil sample (0.004 ppb) is an order of magnitude less than the screening criteria of the ATSDR established screening level of 0.05 ppb (ATSDR, 2008) and almost three orders of magnitude less than the minimum screening criteria of the USEPA draft recommended interim preliminary remediation goal (USEPA, 2010) of 1 ppb for dioxin toxicity equivalents (TEQs) in residential soil. The human health risk assessment for the site identified TEQ as a COPC however TEQ was not a COC for cancer risk for the total residential risk from surface soil. No change will be made based on this comment.

NAVAL TRAINING CENTER
GREAT LAKES, ILLINOIS

BASE MAP

SHOWING CONDITIONS AS OF 31 DEC. 1958

DEPARTMENT OF THE NAVY

GRAPHIC SCALE

SYMBOL	DESCRIPTION	DATE	APPROVAL
	REVISIONS		

DESIGNED
DRAWN
TRACED
CHECKED
QUANTITY
DIRECTOR

APPROVED

Appendix G-Uncertainty - TABLE G-1
IEPA SUGGESTED NON-CANCER TOXICITY DATA - ORAL/DERMAL
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD Used in draft RA		IEPA suggested RfD	Difference between IEPA RfD and Draft RA RfD
		Value	Units		
Chronic Toxicity Criteria					
NAPHTHALENE	Chronic	0.02	mg/kg/day	–	
TCDD	Chronic	1.00E-09	mg/kg/day	2.00E-08	**RA more conservative**
TETRACHLOROETHYLENE	Chronic	1.00E-02	mg/kg/day	–	
ALUMINUM	Chronic	1.0E+00	mg/kg/day	–	
ANTIMONY	Chronic	4.0E-04	mg/kg/day	–	
ARSENIC	Chronic	3.0E-04	mg/kg/day	–	
BARIUM	Chronic	2.0E-01	mg/kg/day	–	
CADMIUM	Chronic	1.0E-03	mg/kg/day	–	
CHROMIUM VI	Chronic	3.0E-03	mg/kg/day	–	
COBALT	Chronic	3.0E-04	mg/kg/day	–	
COPPER	Chronic	4.0E-02	mg/kg/day	–	
IRON	Chronic	7.0E-01	mg/kg/day	–	
LEAD	NA	NA	NA	–	
MANGANESE	Chronic	4.7E-02	mg/kg/day	0.02	**RA less conservative**
MERCURY[1]	Chronic	3.0E-04	mg/kg/day	–	
SELENIUM	Chronic	5.0E-03	mg/kg/day	–	
VANADIUM	Chronic	9.0E-03	mg/kg/day	0.005 [2]	**Based on new RSL. RA less conservative.**
ZINC	Chronic	0.3	mg/kg/day	–	
Subchronic Toxicity Criteria					
ALUMINUM	Subchronic	2.0E+00	mg/kg/day	1 [3]	**RA less conservative**
ARSENIC	Subchronic	5.0E-03	mg/kg/day	0.003	**RA less conservative**
CHROMIUM VI	Subchronic	2.0E-02	mg/kg/day	0.005	**RA less conservative**
MERCURY[1]	Subchronic	3.0E-03	mg/kg/day	–	
NAPHTHALENE	Chron for SubChron	2.0E-02	mg/kg/day	0.6	**ATSDR intermediate RfD. RA more conservative.**
TCDD	Chron for SubChron	1.0E-09	mg/kg/day	2.00E-08	**RA more conservative**
TETRACHLOROETHYLENE	Chron for SubChron	1.0E-02	mg/kg/day	–	
ANTIMONY	Chron for SubChron	4.0E-04	mg/kg/day	0.0004	PPRTV subchronic RfD is the same as IRIS chronic RfD
BARIUM	Chron for SubChron	2.0E-01	mg/kg/day	0.2	ATSDR intermediate RfD. Same as chronic
CADMIUM	Chron for SubChron	1.0E-03	mg/kg/day	0.0005	ATSDR intermediate RfD. Lower than IRIS chronic value; therefore no change.
COBALT	Chron for SubChron	3.0E-04	mg/kg/day	0.003	**PPRTV for subchronic. RA RfD is more conservative**
COPPER	Chron for SubChron	4.0E-02	mg/kg/day	0.01	ATSDR intermediate RfD. Lower than IRIS chronic value; therefore no change.
IRON	Chron for SubChron	7.0E-01	mg/kg/day	0.7	PPRTV subchronic RfD same as chronic RfD.
MANGANESE	Chron for SubChron	4.7E-02	mg/kg/day	0.02	**RA less conservative**
MERCURY[1]	Chron for SubChron	3.0E-03	mg/kg/day	–	
SELENIUM	Chron for SubChron	5.0E-03	mg/kg/day	0.005	HEAST subchronic RfD same as IRIS chronic value.
VANADIUM	Chron for SubChron	9.0E-03	mg/kg/day	0.005	**Based on new RSL. RA less conservative.**
ZINC	Chron for SubChron	3.0E-01	mg/kg/day	0.3	ATSDR intermediate RfD. Same as chronic

Notes:

[1] - Values are for mercuric chloride.

[2] - RfD 0.005 from RSL (Nov. 2011).

[3] - based on IEPA comments on Site 21.

Bold = IEPA-suggested RfDs that differ from those in Site 9 Risk Assessment. Addressed in Uncertainty Section and Appendix.

ATSDR = Agency for Toxic Substances and Disease Registry

PPRTV = Provisional Peer Reviewed Toxicity Value

TABLE 7.1 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF CONSTRUCTION WORKERS TO SUBSURFACE SOIL
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Subsurface Soil
Exposure Point: Entire Site
Receptor Population: Construction Worker
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose (Subchronic[2] If available)	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	BAP EQUIVALENT (FULL DLs)	9.51E-01	mg/kg	9.51E-01	3.2E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	3.80E-01	mg/kg	3.80E-01	1.3E-06	mg/kg-day	2.0E-02	mg/kg-day	NA	NA	6.4E-05
	TCDD TEQs (FULL DLs)	8.92E-06	mg/kg	8.92E-06	3.0E-11	mg/kg-day	1.0E-09	mg/kg-day	NA	NA	3.0E-02
	ALUMINUM	1.97E+04	mg/kg	1.97E+04	6.6E-02	mg/kg-day	*2.0E+00*	mg/kg-day	NA	NA	3.3E-02
	ANTIMONY	1.18E+01	mg/kg	1.18E+01	4.0E-05	mg/kg-day	4.0E-04	mg/kg-day	NA	NA	9.9E-02
	ARSENIC	1.15E+02	mg/kg	1.15E+02	3.9E-04	mg/kg-day	*5.0E-03*	mg/kg-day	NA	NA	7.7E-02
	BARIUM	1.22E+03	mg/kg	1.22E+03	4.1E-03	mg/kg-day	2.0E-01	mg/kg-day	NA	NA	2.1E-02
	CADMIUM	8.04E+00	mg/kg	8.04E+00	2.7E-05	mg/kg-day	1.0E-03	mg/kg-day	NA	NA	2.7E-02
	CHROMIUM	3.15E+01	mg/kg	3.15E+01	1.1E-04	mg/kg-day	*2.0E-02*	mg/kg-day	NA	NA	5.3E-03
	COBALT	2.21E+01	mg/kg	2.21E+01	7.4E-05	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	2.5E-01
	COPPER	1.14E+03	mg/kg	1.14E+03	3.8E-03	mg/kg-day	4.0E-02	mg/kg-day	NA	NA	9.6E-02
	IRON	5.24E+04	mg/kg	5.24E+04	1.8E-01	mg/kg-day	7.0E-01	mg/kg-day	NA	NA	2.5E-01
	MANGANESE	1.09E+03	mg/kg	1.09E+03	3.7E-03	mg/kg-day	4.7E-02	mg/kg-day	NA	NA	7.8E-02
	MERCURY	3.15E+01	mg/kg	3.15E+01	1.1E-04	mg/kg-day	*3.0E-03*	mg/kg-day	NA	NA	3.5E-02
	VANADIUM	3.62E+01	mg/kg	3.62E+01	1.2E-04	mg/kg-day	9.0E-03	mg/kg-day	NA	NA	1.4E-02
	ZINC	7.92E+02	mg/kg	7.92E+02	2.7E-03	mg/kg-day	3.0E-01	mg/kg-day	NA	NA	8.9E-03
	(total)										**1.0E+00**
Dermal	BAP EQUIVALENT (FULL DLs)	9.51E-01	mg/kg	9.51E-01	1.2E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	3.80E-01	mg/kg	3.80E-01		mg/kg-day	2.0E-02	mg/kg-day	NA	NA	
	TCDD TEQs (FULL DLs)	8.92E-06	mg/kg	8.92E-06	2.7E-12	mg/kg-day	1.0E-09	mg/kg-day	NA	NA	2.7E-03
	ALUMINUM	1.97E+04	mg/kg	1.97E+04		mg/kg-day	*2.0E+00*	mg/kg-day	NA	NA	
	ANTIMONY	1.18E+01	mg/kg	1.18E+01		mg/kg-day	4.0E-04	mg/kg-day	NA	NA	
	ARSENIC	1.15E+02	mg/kg	1.15E+02	3.5E-05	mg/kg-day	*5.0E-03*	mg/kg-day	NA	NA	7.0E-03
	BARIUM	1.22E+03	mg/kg	1.22E+03		mg/kg-day	2.0E-01	mg/kg-day	NA	NA	
	CADMIUM	8.04E+00	mg/kg	8.04E+00	8.1E-08	mg/kg-day	2.5E-05	mg/kg-day	NA	NA	3.2E-03
	CHROMIUM	3.15E+01	mg/kg	3.15E+01		mg/kg-day	*2.0E-02*	mg/kg-day	NA	NA	
	COBALT	2.21E+01	mg/kg	2.21E+01		mg/kg-day	3.0E-04	mg/kg-day	NA	NA	
	COPPER	1.14E+03	mg/kg	1.14E+03		mg/kg-day	4.0E-02	mg/kg-day	NA	NA	
	IRON	5.24E+04	mg/kg	5.24E+04		mg/kg-day	7.0E-01	mg/kg-day	NA	NA	
	MANGANESE	1.09E+03	mg/kg	1.09E+03		mg/kg-day	1.4E-01	mg/kg-day	NA	NA	
	MERCURY	3.15E+01	mg/kg	3.15E+01		mg/kg-day	*3.0E-03*	mg/kg-day	NA	NA	
	VANADIUM	3.62E+01	mg/kg	3.62E+01		mg/kg-day	9.0E-03	mg/kg-day	NA	NA	
	ZINC	7.92E+02	mg/kg	7.92E+02		mg/kg-day	3.0E-01	mg/kg-day	NA	NA	
	(total)										**1.3E-02**
								Total Hazard Index Across All Exposure Routes/Pathways			**1.0E+00**

[1] Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

[2] Subchronic values in *italics*.

Dermal Absorption Fraction from Soil(ABS) (USEPA, July 2004):

Dioxins/furans - 0.03 Arsenic - 0.03 Cadmium - 0.001

PAHs - 0.13 Other Metals and Volatiles - not evaluated for dermal contact with soil.

Appendix G-Uncertainty TABLE 7.1 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF CONSTRUCTION WORKERS TO SUBSURFACE SOIL
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Subsurface Soil
Exposure Point: Entire Site
Receptor Population: Construction Worker
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	BAP EQUIVALENT (FULL DLs)	9.51E-01	mg/kg	9.51E-01	3.2E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	3.80E-01	mg/kg	3.80E-01	1.3E-06	mg/kg-day	**6.0E-01**	mg/kg-day	NA	NA	2.1E-06
	TCDD TEQs (FULL DLs)	8.92E-06	mg/kg	8.92E-06	3.0E-11	mg/kg-day	**2.0E-08**	mg/kg-day	NA	NA	1.5E-03
	ALUMINUM	1.97E+04	mg/kg	1.97E+04	6.6E-02	mg/kg-day	**1.0E+00**	mg/kg-day	NA	NA	6.6E-02
	ANTIMONY	1.18E+01	mg/kg	1.18E+01	4.0E-05	mg/kg-day	4.0E-04	mg/kg-day	NA	NA	9.9E-02
	ARSENIC	1.15E+02	mg/kg	1.15E+02	3.9E-04	mg/kg-day	**3.0E-03**	mg/kg-day	NA	NA	1.3E-01
	BARIUM	1.22E+03	mg/kg	1.22E+03	4.1E-03	mg/kg-day	2.0E-01	mg/kg-day	NA	NA	2.1E-02
	CADMIUM	8.04E+00	mg/kg	8.04E+00	2.7E-05	mg/kg-day	1.0E-03	mg/kg-day	NA	NA	2.7E-02
	CHROMIUM	3.15E+01	mg/kg	3.15E+01	1.1E-04	mg/kg-day	**5.0E-03**	mg/kg-day	NA	NA	2.1E-02
	COBALT	2.21E+01	mg/kg	2.21E+01	7.4E-05	mg/kg-day	**3.0E-03**	mg/kg-day	NA	NA	2.5E-02
	COPPER	1.14E+03	mg/kg	1.14E+03	3.8E-03	mg/kg-day	4.0E-02	mg/kg-day	NA	NA	9.6E-02
	IRON	5.24E+04	mg/kg	5.24E+04	1.8E-01	mg/kg-day	7.0E-01	mg/kg-day	NA	NA	2.5E-01
	MANGANESE	1.09E+03	mg/kg	1.09E+03	3.7E-03	mg/kg-day	**2.0E-02**	mg/kg-day	NA	NA	1.8E-01
	MERCURY	3.15E+01	mg/kg	3.15E+01	1.1E-04	mg/kg-day	3.0E-03	mg/kg-day	NA	NA	3.5E-02
	VANADIUM	3.62E+01	mg/kg	3.62E+01	1.2E-04	mg/kg-day	**5.0E-03**	mg/kg-day	NA	NA	2.4E-02
	ZINC	7.92E+02	mg/kg	7.92E+02	2.7E-03	mg/kg-day	3.0E-01	mg/kg-day	NA	NA	8.9E-03
	(total)										9.9E-01
Dermal	BAP EQUIVALENT (FULL DLs)	9.51E-01	mg/kg	9.51E-01	1.2E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	3.80E-01	mg/kg	3.80E-01		mg/kg-day	6.0E-01	mg/kg-day	NA	NA	
	TCDD TEQs (FULL DLs)	8.92E-06	mg/kg	8.92E-06	2.7E-12	mg/kg-day	**2.0E-08**	mg/kg-day	NA	NA	1.4E-04
	ALUMINUM	1.97E+04	mg/kg	1.97E+04		mg/kg-day	1.0E+00	mg/kg-day	NA	NA	
	ANTIMONY	1.18E+01	mg/kg	1.18E+01		mg/kg-day	4.0E-04	mg/kg-day	NA	NA	
	ARSENIC	1.15E+02	mg/kg	1.15E+02	3.5E-05	mg/kg-day	**3.0E-03**	mg/kg-day	NA	NA	1.2E-02
	BARIUM	1.22E+03	mg/kg	1.22E+03		mg/kg-day	2.0E-01	mg/kg-day	NA	NA	
	CADMIUM	8.04E+00	mg/kg	8.04E+00	8.1E-08	mg/kg-day	2.5E-05	mg/kg-day	NA	NA	3.2E-03
	CHROMIUM	3.15E+01	mg/kg	3.15E+01		mg/kg-day	1.3E-04	mg/kg-day	NA	NA	
	COBALT	2.21E+01	mg/kg	2.21E+01		mg/kg-day	3.0E-03	mg/kg-day	NA	NA	
	COPPER	1.14E+03	mg/kg	1.14E+03		mg/kg-day	4.0E-02	mg/kg-day	NA	NA	
	IRON	5.24E+04	mg/kg	5.24E+04		mg/kg-day	7.0E-01	mg/kg-day	NA	NA	
	MANGANESE	1.09E+03	mg/kg	1.09E+03		mg/kg-day	2.0E-02	mg/kg-day	NA	NA	
	MERCURY	3.15E+01	mg/kg	3.15E+01		mg/kg-day	3.0E-03	mg/kg-day	NA	NA	
0.02	VANADIUM	3.62E+01	mg/kg	3.62E+01		mg/kg-day	5.0E-03	mg/kg-day	NA	NA	
	ZINC	7.92E+02	mg/kg	7.92E+02		mg/kg-day	3.0E-01	mg/kg-day	NA	NA	
	(total)										1.5E-02
								Total Hazard Index Across All Exposure Routes/Pathways			1.0E+00

1 Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

Bold text = Proposed toxicity values based on IEPA comments or response to comments.

Dermal Absorption Fraction from Soil(ABS) (USEPA, July 2004):
Dioxins/furans - 0.03 Arsenic - 0.03 Cadmium - 0.001
PAHs - 0.13 Other Metals and Volatiles - not evaluated for dermal contact with soil.

TABLE 7.3 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF CONSTRUCTION WORKERS TO GROUNDWATER
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Entire Site
Receptor Population: Construction Worker
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose (Subchronic (2) if available)	Reference Dose Units	Hazard Quotient
Dermal	BAP EQUIVALENT (FULL DLs)	1.4E-04	mg/L	1.39E-04	mg/L	M	(3)	mg/kg-day		mg/kg-day	NA
	TCDD TEQs (FULL DLs)	1.3E-08	mg/L	1.30E-08	mg/L	M	(3)	mg/kg-day	1.00E-09	mg/kg-day	NA
	ARSENIC	1.3E-02	mg/L	1.34E-02	mg/L	M	1.8E-06	mg/kg-day	*5.00E-03*	mg/kg-day	3.6E-04
	BARIUM	1.7E+00	mg/L	1.69E+00	mg/L	M	2.3E-04	mg/kg-day	1.40E-02	mg/kg-day	1.6E-02
	COBALT	3.3E-03	mg/L	3.30E-03	mg/L	M	4.4E-07	mg/kg-day	3.00E-04	mg/kg-day	1.5E-03
	IRON	1.3E+01	mg/L	1.25E+01	mg/L	M	1.7E-03	mg/kg-day	7.00E-01	mg/kg-day	2.4E-03
	MANGANESE	7.4E-01	mg/L	7.43E-01	mg/L	M	1.0E-04	mg/kg-day	9.60E-04	mg/kg-day	1.0E-01
	SELENIUM	2.3E-02	mg/L	2.30E-02	mg/L	M	3.1E-06	mg/kg-day	5.00E-03	mg/kg-day	6.2E-04
	(total)										**1.3E-01**
											0.1

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

(2) *Subchronic values in italics.*

(3) Chemical with very high log Kow and therefore outside of the predictive range of the EPA dermal uptake model from water.

NA = Not assessed for this pathway.

Appendix G-Uncertainty TABLE 7.3 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF CONSTRUCTION WORKERS TO GROUNDWATER
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Entire Site
Receptor Population: Construction Worker
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose (Subchronic (2) if available)	Reference Concentration Units	Hazard Quotient
Dermal	BAP EQUIVALENT (FULL DLs)	1.4E-04	mg/L	1.39E-04	mg/L	M	(3)	mg/kg-day		NA	NA
	TCDD TEQs (FULL DLs)	1.3E-08	mg/L	1.30E-08	mg/L	M	(3)	mg/kg-day	**2.00E-08**	NA	NA
	ARSENIC	1.3E-02	mg/L	1.34E-02	mg/L	M	1.8E-06	mg/kg-day	***3.00E-03***	NA	6.0E-04
	BARIUM	1.7E+00	mg/L	1.69E+00	mg/L	M	2.3E-04	mg/kg-day	1.40E-02	NA	1.6E-02
	COBALT	3.3E-03	mg/L	3.30E-03	mg/L	M	4.4E-07	mg/kg-day	**3.00E-03**	NA	1.5E-04
	IRON	1.3E+01	mg/L	1.25E+01	mg/L	M	1.7E-03	mg/kg-day	7.00E-01	NA	2.4E-03
	MANGANESE	7.4E-01	mg/L	7.43E-01	mg/L	M	1.0E-04	mg/kg-day	**8.00E-04**	NA	1.3E-01
	SELENIUM	2.3E-02	mg/L	2.30E-02	mg/L	M	3.1E-06	mg/kg-day	5.00E-03	NA	6.2E-04
	(total)										1.45E-01

Total Hazard Index Across All Exposure Routes/Pathways 0.1

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

(2) Subchronic values in *italics*.

(3) Chemical with very high log Kow and therefore outside of the predictive range of the EPA dermal uptake model from water.

NA = Not assessed for this pathway.

Bold text = Proposed toxicity values based on IEPA comments or response to comments.

TABLE 7.9 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF HYPOTHETICAL FUTURE CHILD RESIDENTS TO SOIL
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Entire Site
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	BAP EQUIVALENT (FULL DLs)	3.60E-01	mg/kg	3.60E-01	4.6E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	1.84E-01	mg/kg	1.84E-01	2.3E-06	mg/kg-day	2.0E-02	mg/kg-day	NA	NA	1.2E-04
	TCDD TEQs (FULL DLs)	7.27E-06	mg/kg	7.27E-06	9.3E-11	mg/kg-day	1.0E-09	mg/kg-day	NA	NA	9.3E-02
	ALUMINUM	1.01E+04	mg/kg	1.01E+04	1.3E-01	mg/kg-day	1.0E+00	mg/kg-day	NA	NA	1.3E-01
	ANTIMONY	1.46E+00	mg/kg	1.46E+00	1.9E-05	mg/kg-day	4.0E-04	mg/kg-day	NA	NA	4.7E-02
	ARSENIC	2.92E+01	mg/kg	2.92E+01	3.7E-04	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	1.2E+00
	BARIUM	1.91E+02	mg/kg	1.91E+02	2.4E-03	mg/kg-day	2.0E-01	mg/kg-day	NA	NA	1.2E-02
	CADMIUM	1.41E+00	mg/kg	1.41E+00	1.8E-05	mg/kg-day	1.0E-03	mg/kg-day	NA	NA	1.8E-02
	CHROMIUM	1.76E+01	mg/kg	1.76E+01	2.2E-04	mg/kg-day	3.0E-03	mg/kg-day	NA	NA	7.5E-02
	COBALT	1.13E+01	mg/kg	1.13E+01	1.4E-04	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	4.8E-01
	COPPER	2.57E+02	mg/kg	2.57E+02	3.3E-03	mg/kg-day	4.0E-02	mg/kg-day	NA	NA	8.2E-02
	IRON	2.64E+04	mg/kg	2.64E+04	3.4E-01	mg/kg-day	7.0E-01	mg/kg-day	NA	NA	4.8E-01
	MANGANESE	6.97E+02	mg/kg	6.97E+02	8.9E-03	mg/kg-day	4.7E-02	mg/kg-day	NA	NA	1.9E-01
	MERCURY	9.70E+00	mg/kg	9.70E+00	1.2E-04	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	4.1E-01
	VANADIUM	2.35E+01	mg/kg	2.35E+01	3.0E-04	mg/kg-day	9.0E-03	mg/kg-day	NA	NA	3.3E-02
	ZINC	2.43E+02	mg/kg	2.43E+02	3.1E-03	mg/kg-day	3.0E-01	mg/kg-day	NA	NA	1.0E-02
	(total)										**3.3E+00**
Dermal	BAP EQUIVALENT (FULL DLs)	3.60E-01	mg/kg	3.60E-01	1.7E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	1.84E-01	mg/kg	1.84E-01		mg/kg-day		mg/kg-day	NA	NA	
	TCDD TEQs (FULL DLs)	7.27E-06	mg/kg	7.27E-06	7.8E-12	mg/kg-day	1.0E-09	mg/kg-day	NA	NA	7.8E-03
	ALUMINUM	1.01E+04	mg/kg	1.01E+04		mg/kg-day		mg/kg-day	NA	NA	
	ANTIMONY	1.46E+00	mg/kg	1.46E+00		mg/kg-day		mg/kg-day	NA	NA	
	ARSENIC	2.92E+01	mg/kg	2.92E+01	3.1E-05	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	1.0E-01
	BARIUM	1.91E+02	mg/kg	1.91E+02		mg/kg-day		mg/kg-day	NA	NA	
	CADMIUM	1.41E+00	mg/kg	1.41E+00	5.0E-08	mg/kg-day	2.5E-05	mg/kg-day	NA	NA	2.0E-03
	CHROMIUM	1.76E+01	mg/kg	1.76E+01		mg/kg-day		mg/kg-day	NA	NA	
	COBALT	1.13E+01	mg/kg	1.13E+01		mg/kg-day		mg/kg-day	NA	NA	
	COPPER	2.57E+02	mg/kg	2.57E+02		mg/kg-day		mg/kg-day	NA	NA	
	IRON	2.64E+04	mg/kg	2.64E+04		mg/kg-day		mg/kg-day	NA	NA	
	MANGANESE	6.97E+02	mg/kg	6.97E+02		mg/kg-day		mg/kg-day	NA	NA	
	MERCURY	9.70E+00	mg/kg	9.70E+00		mg/kg-day		mg/kg-day	NA	NA	
	VANADIUM	2.35E+01	mg/kg	2.35E+01		mg/kg-day		mg/kg-day	NA	NA	
	ZINC	2.43E+02	mg/kg	2.43E+02		mg/kg-day		mg/kg-day	NA	NA	
	(total)										**1.1E-01**
								Total Hazard Index Across All Exposure Routes/Pathways			**3.4E+00**

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

Dermal Absorption Fraction from Soil(ABS) (USEPA, July 2004):
Dioxins/furans - 0.03 Arsenic - 0.03 Cadmium - 0.001
PAHs - 0.13 Other Metals and Volatiles - not evaluated for dermal contact with soil.

Appendix G-Uncertainty TABLE 7.9 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF HYPOTHETICAL FUTURE CHILD RESIDENTS TO SOIL
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Entire Site
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	BAP EQUIVALENT (FULL DLs)	3.60E-01	mg/kg	3.60E-01	4.6E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	1.84E-01	mg/kg	1.84E-01	2.3E-06	mg/kg-day	2.0E-02	mg/kg-day	NA	NA	1.2E-04
	TCDD TEQs (FULL DLs)	7.27E-06	mg/kg	7.27E-06	9.3E-11	mg/kg-day	**2.0E-08**	mg/kg-day	NA	NA	4.6E-03
	ALUMINUM	1.01E+04	mg/kg	1.01E+04	1.3E-01	mg/kg-day	1.0E+00	mg/kg-day	NA	NA	1.3E-01
	ANTIMONY	1.46E+00	mg/kg	1.46E+00	1.9E-05	mg/kg-day	4.0E-04	mg/kg-day	NA	NA	4.7E-02
	ARSENIC	2.92E+01	mg/kg	2.92E+01	3.7E-04	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	1.2E+00
	BARIUM	1.91E+02	mg/kg	1.91E+02	2.4E-03	mg/kg-day	2.0E-01	mg/kg-day	NA	NA	1.2E-02
	CADMIUM	1.41E+00	mg/kg	1.41E+00	1.8E-05	mg/kg-day	1.0E-03	mg/kg-day	NA	NA	1.8E-02
	CHROMIUM	1.76E+01	mg/kg	1.76E+01	2.2E-04	mg/kg-day	3.0E-03	mg/kg-day	NA	NA	7.5E-02
	COBALT	1.13E+01	mg/kg	1.13E+01	1.4E-04	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	4.8E-01
	COPPER	2.57E+02	mg/kg	2.57E+02	3.3E-03	mg/kg-day	4.0E-02	mg/kg-day	NA	NA	8.2E-02
	IRON	2.64E+04	mg/kg	2.64E+04	3.4E-01	mg/kg-day	7.0E-01	mg/kg-day	NA	NA	4.8E-01
	MANGANESE	6.97E+02	mg/kg	6.97E+02	8.9E-03	mg/kg-day	**2.0E-02**	mg/kg-day	NA	NA	4.5E-01
	MERCURY	9.70E+00	mg/kg	9.70E+00	1.2E-04	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	4.1E-01
	VANADIUM	2.35E+01	mg/kg	2.35E+01	3.0E-04	mg/kg-day	**5.0E-03**	mg/kg-day	NA	NA	6.0E-02
	ZINC	2.43E+02	mg/kg	2.43E+02	3.1E-03	mg/kg-day	3.0E-01	mg/kg-day	NA	NA	1.0E-02
	(total)										3.5E+00
Dermal	BAP EQUIVALENT (FULL DLs)	3.60E-01	mg/kg	3.60E-01	1.7E-06	mg/kg-day		mg/kg-day	NA	NA	
	NAPHTHALENE	1.84E-01	mg/kg	1.84E-01		mg/kg-day		mg/kg-day	NA	NA	
	TCDD TEQs (FULL DLs)	7.27E-06	mg/kg	7.27E-06	7.8E-12	mg/kg-day	**2.0E-08**	mg/kg-day	NA	NA	3.9E-04
	ALUMINUM	1.01E+04	mg/kg	1.01E+04		mg/kg-day		mg/kg-day	NA	NA	
	ANTIMONY	1.46E+00	mg/kg	1.46E+00		mg/kg-day		mg/kg-day	NA	NA	
	ARSENIC	2.92E+01	mg/kg	2.92E+01	3.1E-05	mg/kg-day	3.0E-04	mg/kg-day	NA	NA	1.0E-01
	BARIUM	1.91E+02	mg/kg	1.91E+02		mg/kg-day		mg/kg-day	NA	NA	
	CADMIUM	1.41E+00	mg/kg	1.41E+00	5.0E-08	mg/kg-day	2.5E-05	mg/kg-day	NA	NA	2.0E-03
	CHROMIUM	1.76E+01	mg/kg	1.76E+01		mg/kg-day		mg/kg-day	NA	NA	
	COBALT	1.13E+01	mg/kg	1.13E+01		mg/kg-day		mg/kg-day	NA	NA	
	COPPER	2.57E+02	mg/kg	2.57E+02		mg/kg-day		mg/kg-day	NA	NA	
	IRON	2.64E+04	mg/kg	2.64E+04		mg/kg-day		mg/kg-day	NA	NA	
	MANGANESE	6.97E+02	mg/kg	6.97E+02		mg/kg-day		mg/kg-day	NA	NA	
	MERCURY	9.70E+00	mg/kg	9.70E+00		mg/kg-day		mg/kg-day	NA	NA	
	VANADIUM	2.35E+01	mg/kg	2.35E+01		mg/kg-day		mg/kg-day	NA	NA	
	ZINC	2.43E+02	mg/kg	2.43E+02		mg/kg-day		mg/kg-day	NA	NA	
	(total)										1.1E-01
										Total Hazard Index Across All Exposure Routes/Pathways	3.6E+00

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

Dermal Absorption Fraction from Soil(ABS) (USEPA, July 2004):
Dioxins/furans - 0.03 Arsenic - 0.03 Cadmium - 0.001
PAHs - 0.13 Other Metals and Volatiles - not evaluated for dermal contact with soil.

Bold text = Proposed toxicity values based on IEPA comments or response to comments.

TABLE 7.11 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF CHILD RESIDENTS TO GROUNDWATER
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Entire Site
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Hazard Quotient
Ingestion	BAP EQUIVALENT (FULL DLs)	1.4E-04	mg/L	1.39E-04	mg/L	M	6.1E-06	mg/kg-day		mg/kg-day	NA
	TCDD TEQs (FULL DLs)	1.3E-08	mg/L	1.30E-08	mg/L	M	1.2E-09	mg/kg-day	1.00E-09	mg/kg-day	1.2E+00
	ARSENIC	1.3E-02	mg/L	1.34E-02	mg/L	M	1.3E-03	mg/kg-day	3.00E-04	mg/kg-day	4.3E+00
	BARIUM	1.7E+00	mg/L	1.69E+00	mg/L	M	1.6E-01	mg/kg-day	2.00E-01	mg/kg-day	8.1E-01
	COBALT	3.3E-03	mg/L	3.30E-03	mg/L	M	3.2E-04	mg/kg-day	3.0E-04	mg/kg-day	1.1E+00
	IRON	1.3E+01	mg/L	1.25E+01	mg/L	M	1.2E+00	mg/kg-day	7.0E-01	mg/kg-day	1.7E+00
	MANGANESE	7.4E-01	mg/L	7.43E-01	mg/L	M	7.1E-02	mg/kg-day	4.70E-02	mg/kg-day	1.5E+00
	SELENIUM	2.3E-02	mg/L	2.30E-02	mg/L	M	2.2E-03	mg/kg-day	5.00E-03	mg/kg-day	4.4E-01
	(total)										11
Dermal	BAP EQUIVALENT	1.4E-04	mg/L	1.39E-04	mg/L	M	(2)	mg/kg-day		mg/kg-day	NA
	TCDD TEQs (FULL DLs)	1.3E-08	mg/L	1.30E-08	mg/L	M	(2)	mg/kg-day	1.00E-09	mg/kg-day	NA
	ARSENIC	1.3E-02	mg/L	1.34E-02	mg/L	M	1.9E-06	mg/kg-day	3.00E-04	mg/kg-day	6.3E-03
	BARIUM	1.7E+00	mg/L	1.69E+00	mg/L	M	2.4E-04	mg/kg-day	1.40E-02	mg/kg-day	1.7E-02
	COBALT	3.3E-03	mg/L	3.30E-03	mg/L	M	4.6E-07	mg/kg-day	3.00E-04	mg/kg-day	1.5E-03
	IRON	1.3E+01	mg/L	1.25E+01	mg/L	M	1.8E-03	mg/kg-day	7.00E-01	mg/kg-day	2.5E-03
	MANGANESE	7.4E-01	mg/L	7.43E-01	mg/L	M	1.0E-04	mg/kg-day	1.88E-03	mg/kg-day	5.6E-02
	SELENIUM	2.3E-02	mg/L	2.30E-02	mg/L	M	3.2E-06	mg/kg-day	5.00E-03	mg/kg-day	6.5E-04
	(total)										0.08
											11

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

(2) Chemical with very high log Kow and therefore outside of the predictive range of the EPA dermal uptake model from water.

NA = Not assessed for this pathway.

Appendix G-Uncertainty TABLE 7.11 - REASONABLE MAXIMUM EXPOSURE (RME)
CALCULATION OF NON-CANCER HAZARDS FROM EXPOSURE OF CHILD RESIDENTS TO GROUNDWATER
SITE 9 - CAMP MOFFETT
NAVAL STATION GREAT LAKES, ILLINOIS

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Entire Site
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Hazard Quotient
Ingestion	BAP EQUIVALENT (FULL DLs)	1.4E-04	mg/L	1.39E-04	mg/L	M	6.1E-06	mg/kg-day		mg/kg-day	NA
	TCDD TEQs (FULL DLs)	1.3E-08	mg/L	1.30E-08	mg/L	M	1.2E-09	mg/kg-day	**2.00E-08**	mg/kg-day	6.2E-02
	ARSENIC	1.3E-02	mg/L	1.34E-02	mg/L	M	1.3E-03	mg/kg-day	3.00E-04	mg/kg-day	4.3E+00
	BARIUM	1.7E+00	mg/L	1.69E+00	mg/L	M	1.6E-01	mg/kg-day	2.00E-01	mg/kg-day	8.1E-01
	COBALT	3.3E-03	mg/L	3.30E-03	mg/L	M	3.2E-04	mg/kg-day	3.0E-04	mg/kg-day	1.1E+00
	IRON	1.3E+01	mg/L	1.25E+01	mg/L	M	1.2E+00	mg/kg-day	7.0E-01	mg/kg-day	1.7E+00
	MANGANESE	7.4E-01	mg/L	7.43E-01	mg/L	M	7.1E-02	mg/kg-day	**2.00E-02**	mg/kg-day	3.6E+00
	SELENIUM	2.3E-02	mg/L	2.30E-02	mg/L	M	2.2E-03	mg/kg-day	5.00E-03	mg/kg-day	4.4E-01
	(total)										12
Dermal	BAP EQUIVALENT	1.4E-04	mg/L	1.39E-04	mg/L	M	(2)	mg/kg-day		mg/kg-day	NA
	TCDD TEQs (FULL DLs)	1.3E-08	mg/L	1.30E-08	mg/L	M	(2)	mg/kg-day	**2.00E-08**	mg/kg-day	NA
	ARSENIC	1.3E-02	mg/L	1.34E-02	mg/L	M	1.9E-06	mg/kg-day	3.00E-04	mg/kg-day	6.3E-03
	BARIUM	1.7E+00	mg/L	1.69E+00	mg/L	M	2.4E-04	mg/kg-day	1.40E-02	mg/kg-day	1.7E-02
	COBALT	3.3E-03	mg/L	3.30E-03	mg/L	M	4.6E-07	mg/kg-day	3.00E-04	mg/kg-day	1.5E-03
	IRON	1.3E+01	mg/L	1.25E+01	mg/L	M	1.8E-03	mg/kg-day	7.00E-01	mg/kg-day	2.5E-03
	MANGANESE	7.4E-01	mg/L	7.43E-01	mg/L	M	1.0E-04	mg/kg-day	**8.00E-04**	mg/kg-day	1.3E-01
	SELENIUM	2.3E-02	mg/L	2.30E-02	mg/L	M	3.2E-06	mg/kg-day	5.00E-03	mg/kg-day	6.5E-04
	(total)										0.16
											12

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

(2) Chemical with very high log Kow and therefore outside of the predictive range of the EPA dermal uptake model from water.

NA = Not assessed for this pathway.

Bold text = Proposed toxicity values based on IEPA comments or response to comments.

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Derivation of Acute and Subchronic Oral Reference Doses for Inorganic Arsenic

August 2002

This document was written by Dr. Robert Benson, US EPA Region VIII. The effort was aided by an EPA/ATSDR Interagency Work Group. The members from EPA included Drs. Robert Benson, Mark Johnson, Roseanne Lorenzana, Mark Maddaloni, Chris Weis, Peter Grevatt and Mr. Mike Beringer. The members from ATSDR included Drs. Selene Chou, David Mellard, and Allan Susten. Three scientists conducted an external peer review of the document on two separate occasions.

Introduction

Arsenic is a naturally occurring element and is usually present at low levels in soil, water, food, and air. There are, however, areas in the world where inorganic arsenic is found at elevated concentrations in environmental media and is associated with adverse health outcomes in these areas. In some cases natural conditions cause these elevated concentrations, in others human activity caused the elevated concentrations. Arsenic has been used in the past in a number of medicines and pesticides and is still sometimes found in herbal and other folk-medicine products.

EPA has established a chronic reference dose (RfD) for inorganic arsenic of 0.0003 mg/kg-day (EPA, 2002), which EPA considers to apply to lifetime exposure. Application of the RfD is not necessarily appropriate for an exposure of much shorter duration. Accordingly, there is a need for reference doses to apply to other exposure situations. In this document acute exposure is defined as an exposure of one to 14 days. Subchronic exposure is defined as an exposure of 15 days to 7 years (approximately 10% of the average human lifespan). Chronic exposure is defined as an exposure of greater than 7 years.

Published literature on the adverse health effects of inorganic arsenic reasonably close to the exposure durations defined above were reviewed. This paper summarizes only those publications that provide specific information on duration of exposure and provide sufficient information to estimate the daily exposure.

Hazard Identification for Inorganic Arsenic (non-cancer endpoints)

Arsenic interferes with the action of enzymes, essential cations, and transcriptional events in cells throughout the body. A multitude of non-cancer effects ensue (see for example, Polson and Tattersall, 1969; National Research Council, 1999; Saha et al., 1999). This discussion focuses on the non-cancer effects from less than chronic exposure as presented in a review (National Research Council, 1999). The reader should consult that source for references to the original scientific literature. Because laboratory animals appear to be less sensitive than humans to the adverse effects of arsenic, the discussion focuses on reports of adverse health effects studies in human populations exposed to inorganic arsenic.

Gastrointestinal Effects

An exposure greater than several milligrams per day usually induces overt gastrointestinal disturbances. The effects range from mild abdominal cramping and diarrhea to severe life-threatening hemorrhagic gastroenteritis associated with shock. Hepatic enlargement is sometimes observed. Mild-to-moderate hepatocellular necrosis, evidenced by increases in serum transaminase, might occur. These effects are reversible. Non-cirrhotic portal hypertension is an uncommon but relatively specific gastrointestinal manifestation associated with exposure from medications or drinking water.

Neurological Effects

An intoxication that produces overt gastrointestinal or cardiovascular symptoms can be followed by the delayed onset of central or peripheral nervous system effects. The central nervous system effects, appearing within 1-5 days, can range from headache and mild confusion to florid

encephalopathy, seizures, and coma. Evidence of peripheral neuropathy, a more common finding, emerges within 1-4 weeks. Histopathological examination of the peripheral nerves is consistent with a sensorimotor axonopathy, although electro-physiological testing can sometimes suggest segmental demyelination. Long term exposure at lower levels may result in subclinical or overt peripheral neuropathy without a previous history of gastrointestinal or cardiovascular signs. Prominent peripheral neuropathy has been reported following ingestion of drinking water containing arsenic at a concentration of 10 mg/L or more. The occurrence of peripheral neuropathy is inconsistent in individuals following exposure to arsenic in drinking water at concentrations of 0.1 - 1.0 mg/L.

Cardiovascular Effects

An exposure in the range of milligrams to grams per day induces the rapid appearance of serious overt cardiovascular manifestations, including hypotension, congestive heart failure, and cardiac arrhythmias. The latter are often preceded by electrocardiographic prolongation of the Q-T interval, occasionally leading to polymorphic ventricular tachycardia. These effects are reversible. Longer term ingestion of inorganic arsenic has been associated with the development of cardiac and peripheral vascular disease, including arterial spasms in the fingers and toes (Raynaud's Syndrome) and thromboangiitis obliterans. Following chronic exposure a severe manifestation of peripheral vascular deficiency results in gangrene of the extremities, particularly the feet (Blackfoot Disease).

Hematological Effects

High dose, short term exposure might result in anemia, leukopenia, and thrombocytopenia. Effects on those cell lineages can be simultaneous and can appear within a week of exposure. The anemia, a consequence of hemolysis or marrow suppression, might be normocytic or megaloblastic. The marrow can also reveal erythroid hyperplasia. Leukopenia can be characterized by neutropenia or lymphopenia. In practically all cases, the hematological abnormalities are reversible; normalization of most cell lineages occurs within weeks of termination of exposure.

Pulmonary Effects

The possible role of ingested arsenic in the genesis of nonmalignant pulmonary disease has been suggested in a few case series. These effects, occurring in individuals with cutaneous lesions, include chronic cough, slight pulmonary fibrosis, and restrictive and obstructive lung disease.

Reproductive and Developmental Effects

Very few studies have been conducted on arsenic and reproductive success in humans and nothing conclusive can be stated from these studies. In studies with laboratory animals, inorganic arsenic has been shown to be a developmental toxicant following i.p. or i.v. administration. A peer review panel evaluated the published data and two unpublished manuscripts to determine whether ingestion or inhalation of inorganic arsenic causes structural malformations (TERA, 1999). The manuscripts have since been published (DeSesso et al., 1998; Holson et al., 2000). TERA stated:

> "The review panel concluded that the existing human epidemiology studies were insufficient to make a determination on the ability of inorganic arsenic to induce malformations in humans, due to weaknesses in characterization of exposures, lack of reporting of birth related outcomes, and limited control of confounders. The panel concluded, based on the new regulatory guideline-compliant studies on arsenic trioxide and arsenic acid in mice, rats, and rabbits, that repeated

oral and inhalation exposures to these forms of inorganic arsenic did not induce structural malformations; even at doses that elicited frank maternal toxicity and lethality."

Endocrine Effects

Two reports have documented an increased prevalence of diabetes mellitus among people also showing skin lesions (keratoses) from areas of Bangladesh (Rahman et al., 1998) and Taiwan (Lai et al., 1994) where the drinking water contains inorganic arsenic. This effect is associated with chronic exposure to inorganic arsenic. There are no reports of diabetes mellitus in populations following acute or subchronic exposure to inorganic arsenic.

Immunological Effects

Immunomodulating and immunotoxic effects of inorganic arsenic have been demonstrated in several experimental models *in vitro* using human and bovine peripheral lymphocytes. Some evidence suggests that the immunosuppressant effects of arsenic contributed to the clinical effectiveness of Fowler's solution and a common side effect of its use, the development of herpes zoster. Several poisoning incidents involving arsenic have been associated with an increase in the prevalence of herpes labialis and herpes zoster in the exposed population.

Cutaneous Effects

In contrast to the many non-specific signs and symptoms that are challenging to diagnose, the classic cutaneous lesions caused by inorganic arsenic are distinctive, characteristic, and appear to be the most sensitive effect due to exposure to inorganic arsenic. Their appearance usually follows a temporal progression, beginning with hyperpigmentation which can occur several weeks after exposure, then progressing to palmar-plantar hyperkeratoses. Although cutaneous manifestations have been most commonly reported following ingestion of arsenic containing drinking water, cohorts exposed to medicinals, contaminated grape beverages, and via inhalation have also shown an increased prevalence of skin lesions. The hyperpigmentation appears in a finely freckled, "raindrop" pattern that is particularly pronounced on the trunk and extremities. In some cases hyperpigmentation also involves mucous membranes such as the tongue or buccal mucosa. The hyperkeratoses appear predominately on the palms and the plantar aspects of the feet. In some cases the hyperkeratoses also appear on the dorsum of the extremities and the trunk. Some evidence suggests that the skin lesions might lessen in severity or regresses when exposure is stopped, but there is no definitive proof that the lesions completely disappear.

Exposure-Response Assessment

The exposures presented in this section are to inorganic arsenic compounds, but are quantified as elemental arsenic. Any assumptions necessary to calculate the exposure are presented in the discussion of each report. The reports are presented in order of increasing duration of exposure.

Acute Exposure

Mizuta et al. (1956) reported a poisoning incident involving the presence of arsenic in soy-sauce. The duration of exposure was 2-3 weeks. Based on the measured concentration of inorganic arsenic in the soy sauce (100 mg/L), the authors estimated that an average of 3 mg of arsenic had been consumed daily throughout this incident. Further details of the exposure reconstruction and

the range of exposure among the affected individuals were not reported. The report provided observations on only selected patients. The selection criteria were not specified. The authors reported clinical observations on 220 subjects (age not specified) out of a total of 417 cases. The patients presented with multifaceted gastrointestinal symptoms, liver enlargement, upper respiratory symptoms, peripheral neuropathy, and skin disorders. An early feature of arsenic toxicity in this cohort was the appearance of facial edema that was most marked on the eyelids. In the majority of patients, the symptoms appeared within two days of ingestion of the contaminated soy sauce and declined even with continued exposure. There was evidence of minor gastrointestinal bleeding (occult blood in gastric juice in 6 of 6 patients reported on and in duodenal juice in 5 of 7 patients reported on). There were abnormalities in electrocardiograms (altered Q-T interval and P- and T-waves) in 16 of 20 patients reported on. These changes were not evident on reexamination after the recovery from the clinical symptoms. An abnormal patellar reflex was evident in 1 of 10 patients reported on during the first week and in more than 50% of 110 patients reported on during the second through the fifth week. It is not clear from the paper if repeated measurements were made on the same patient. The patellar reflex did not return to normal even after exposure to arsenic ceased. The concentration of urinary arsenic was reported for five patients. In two patients 5 days after exposure ceased, the concentration of urinary arsenic was 1 and 8.85 mg/L. In the other three patients 9 to 10 days after exposure ceased, the concentration of urinary arsenic was 1, 1.4, and 2 mg/L. As the majority of ingested arsenic is excreted in urine within 72 hours after exposure (Casarett and Doull, 1996), these results suggest an exposure greater than the 3 mg/day estimated by the authors or a continuing source of exposure to inorganic arsenic. Based on the concentration of arsenic in the contaminated soy-sauce (100 mg/L), the authors estimated the average exposure at 3 mg/day. The average body weight was not reported. EPA assumes an average body weight of 55 kg in this Asian population. [This assumption is consistent with the body weight used for establishing the arsenic RfD (EPA, 2002)]. The estimated exposure in this incident is 0.05 mg/kg-day.

Franzblau and Lilis (1989) describe two cases (a wife and husband) of arsenic intoxication from well water containing arsenic (chemical form not specified). The total inorganic arsenic concentration of the well ranged from 9 to 10.9 mg/L. Visits to the home occurred once or twice each week for two months. The symptoms worsened when the couple moved into the house and increased their consumption of well water. Both individuals then visited their physician and were admitted to a hospital. The woman reported that immediately after consumption of the well water at the house began, the patients experienced gastrointestinal symptoms (occasional nausea, diarrhea, abdominal cramps). With continued exposure, the symptoms worsened and the woman reported occasional vomiting, paresthesia, and a sensation of swelling and irritation of the eyes and sinuses. It is not clear from the paper when these latter symptoms appeared during this incident. The woman (body weight not stated) reported that she consumed 1-2 glasses of water from the contaminated well per visit to the home. No exposure information is provided for the man. The estimated consumption of water is 0.238 - 0.475 L/day [1-2 glasses/day x 8 ounces/glass x 3.8 L/128 ounces]. EPA assumes a body weight of 65 kg. On the days when the contaminated water was consumed, the estimated exposure is 0.03 - 0.08 mg/kg-day [0.238 - 0.475 L/day x 9 - 10.9 mg/L x 1/65 kg].

Arsenic trioxide has been found to induce remission in patients with acute promyelocytic

leukemia. The clinical response is associated with incomplete cytodifferentiation and the induction of apoptosis with caspase activation in leukemic cells (Soignet et al., 1998). Although exposure to arsenic in this clinical setting is by i.v. infusion, typically for about 30 days, and these individuals have severely compromised health status, this information is included in the document because it provides useful information on the exposure-response relationship for the cardiac and neurological effects sometimes reported following oral exposure to inorganic arsenic (Foy et al., 1992; Franzblau and Lilis, 1989; Mizuta et al., 1956; Silver and Wainman, 1952; Wagner, 1979). In addition, the patients in these clinical trials were administered a known and controlled dose of inorganic arsenic and their health status was closely monitored.

Abnormalities in electrocardiography have frequently been reported in clinical trials using i.v. infusion of arsenic at 0.12 mg/kg-day (Barbey et al., 2001; Soignet et al., 2001; Wang, 2001). The abnormality usually is Long QT Syndrome. In a few cases, Torsades de Pointes has been observed with a fatal outcome. Niu et al. (1999) and Chen et al. (2001) report the incidence of side effects in 58 patients administered arsenic at 0.12 mg/kg-day and 20 patients administered arsenic at 0.06 mg/kg-day. At the high dose, the incidence of adverse side reactions was: skin reaction (15/58), gastrointestinal disturbance (14/50), cardiac dysfunction (9/58), facial edema and neuropathy (5/58), and liver dysfunction (22/58, with two fatalities from liver failure). At the low dose, the incidence of adverse side reactions was: skin reaction (2/20) and liver dysfunction (4/20), but no cases of cardiac dysfunction or facial edema or neuropathy. The skin reactions (rash, itching, erythema) and liver dysfunction (elevated aspartate transaminase and alanine transaminase) were of a mild nature. Soignet et al. (2001) report peripheral neuropathy in 17 of 40 patients at a dose of arsenic of 0.11 mg/kg-day. The majority of patients reported only mild symptoms. The symptoms resolved after treatment stopped. Soignet et al. (2001) evaluated liver function but made no mention of liver dysfunction in the report.

These data from acute oral exposure are summarized in the table below. The data support the conclusion that exposure to inorganic arsenic at 0.05 mg/kg-day for one to two days will cause adverse effects.

Exposure quantified	Duration	Effects observed	Cases	Exposure (mg/kg-day)	Reference
Soy sauce	2 days to 3 weeks	edema, cardiac, gastrointestinal, respiratory, neurological, skin	417	0.05	Mizuta et al., 1956
Drinking water	1-2 days per week for two months	gastrointestinal, neurological	2	0.03 - 0.08 intermittent	Franzblau and Lilis, 1989

Subchronic Exposure

Wagner et al. (1979) describe a case of arsenic intoxication arising from well water containing 1.2 mg/L of arsenic. The duration of exposure was 4 months. The patient originally presented in July 1962 with a one month history of symptoms of paresthesia, skin lesions (cracking and scaling of palmar surfaces), weakness, nausea, vomiting, diarrhea, substantial weight loss (18 kg), and alopecia. The patient and her husband used the well water for all household purposes. The patient (body weight not reported) admitted to being a heavy coffee drinker, conservatively estimating her intake at 12 to 14 cups per day. The estimated consumption of well water from coffee is 2.7 L/day (average) [12-14 cups/day x 6-8 ounces/cup x 3.8L/128 ounces]. Other consumption of well water is not mentioned. The total consumption of well water is likely >2.7 L/day. After a latent period of 14 years, multiple cutaneous carcinomas developed. Histopathological examination revealed two types of lesions (*in situ* squamous cell carcinoma and superficial multicentric basal cell carcinomas). It is not clear from the paper whether she continued to drink water from the well containing arsenic during the intervening 14 years. EPA assumes a body weight of 65 kg. This value, however, is uncertain because of the profound weight loss reported by the patient. The estimated exposure is >0.05 mg/kg-day [>2.7 L/day x 1.2 mg/L x 1/65 kg].

Several papers describe case reports of toxicity in adults following the medicinal use of Fowler's solution (arsenic trioxide dissolved in potassium bicarbonate). Wade and Fraser (1953) reported on one individual who was treated for 15 months and received a total of 80 grains of arsenic trioxide [equivalent to 3900 mg of arsenic; 80 grains x 65 mg/grain x 149.84/197.84]. He developed pigmentation of the skin on the trunk and thighs, hyperkeratosis of the palms, and an enlarged liver. His body weight is not reported; EPA assumes 70 kg. The estimated exposure is 0.12 mg/kg-day [3900 mg x 1/(15 months x 30 days/month) x 1/70 kg]. Silver and Wainman (1952) report on one patient who was treated for 28 months. The signs of arsenic poisoning included freckling and darkening of the nipples after 13 months; redness and puffiness about the eyes and hyperkeratosis after 18 months; and paresthesia and weakness after two years. She took 2 to 4 teaspoons daily of a solution containing 0.44 mg arsenic trioxide/ml. Her body weight is not provided; EPA assumes 65 kg. Her estimated exposure is 0.05 - 0.10 mg/kg-day [0.44 mg/ml x 2-4 tsp/day x 5 ml/tsp x 149.84/197.84 x 1/65 kg]. Morris et al. (1974) provided a case report of an individual who was treated for three years. The patient had skin pigmentation and non-cirrhotic portal hypertension. He received a total of 6.5 grams of arsenic. The body weight is not provided; EPA assumes 70 kg. The estimated exposure is 0.08 mg/kg-day [6500 mg x 1/(3 yr x 365 days/yr) x 1/70 kg].

Foy et al. (1992) report on four cases of children from a tin mining area of Thailand whose drinking water contained arsenic. The arsenic concentration in shallow wells varied between 0.02 and 2.7 mg/L. The duration of exposure was three to six years. Each child showed skin lesions typical of arsenic intoxication. One child (described below) also showed neurological and liver effects. Specific information on the arsenic concentration of the drinking water was supplied in only one case. A nine-year-old girl had been living in the area for six years and had been drinking water from a well containing arsenic at 2.7 mg/L. She had hyperkeratosis of both palms and soles, and generalized pigmentation of the feet. She had developed weakness three years previously, and had anorexia and chronic cough for one year. She also had weakness of her wrist joints and an enlarged liver. Her body weight and

drinking water consumption are not provided. EPA assumes a consumption of 0.035 L/kg-day (EPA, 1997, Table 3-30). The estimated exposure is an underestimate as there is no allowance for increased water consumption in this tropical area. Her estimated exposure is >0.09 mg/kg-day [2.7 mg/L x 0.035 L/kg-day].

Huang et al. (1985) report an investigation of endemic arsenicism in Kuitun area, Xinjiang, China. The water supplying the population came from a deep artesian well with 0.6 mg/L of arsenic. The well was first used in 1969. In 1982 the authors examined 336 individuals. There was no control group. One-hundred and fifty people (44.6%) showed dyspigmentation (diffuse brownish pigmented macules and spots mixed with depigmented areas) and hyperkeratosis chiefly on the palms and soles. The duration of exposure ranged from six months to 12 years. Because no other symptoms presented with the cutaneous lesions, most patients failed to remember the exact time of onset of symptoms. The authors stated that individuals drank more than two liters of water daily with the highest intake as eight liters daily. EPA assumes an average consumption of drinking water of 5 L/day. No information is provided on body weight. EPA assumes a body weight of 55 kg for this Asian population (EPA, 2002). The estimated exposure is 0.05 mg/kg-day [0.6 x 5 L/day x 1/55 kg]. The authors also reported that discontinuing drinking the contaminated water distinctly lessened the dyspigmentation and some individuals normalized after one year. Palmar and plantar keratosis disappeared more slowly. In some individuals keratotic lesions continued to develop. The authors did not make it clear if the keratotic lesions continued to appear in previously asymptomatic or symptomatic individuals.

A series of papers describe the arsenic poisoning in Antofagasta, Chile (Borgono and Greiber, 1972; Borgono et al., 1977; Zaldivar, 1974; Zaldivar, 1977; Zaldivar and Guillier, 1977; Zaldivar and Ghai, 1980a, 1980b). The population of the region was exposed to arsenic from the public water supply and from food. Zaldivar and Guillier (1977) estimated the number of persons exposed at 265,000, including 106,000 infants (0-12 months) and children (1-15 years). Of particular interest are the reports of skin lesions (leuko-melanoderma and/or hyperkeratosis of palms and soles, sometimes accompanied by scaling of the skin) in children. Exposure to drinking water containing arsenic at a mean concentration of 0.6 mg/L began in 1958. Children exhibiting skin lesions began to appear in 1962 (Borgono and Greiber, 1972; Zaldivar, 1974). The prevalence of skin lesions in 1968-1969 was reported in two independent studies. Borgono and Greiber (1972) report a prevalence of 12% in 27,088 in school age children. Zaldivar and Ghai (1980b) report a prevalence of 12.3% in 300 children whose age ranged from birth to 15. The children in both investigations were exposed to arsenic in the drinking water from birth. The calculated mean exposure for a child 0-10 years old was reported as 0.0633 mg/kg-day (Zaldivar, 1977; Zaldivar and Ghai, 1980a, 1980b). This value was determined using the average measured concentration of arsenic in drinking water, the measured content of arsenic in a variety of foods, and the average body weight of the children (Zaldivar, 1977; Zaldivar and Ghai, 1980a, 1980b). There were also reports of five deaths attributed at least in part to exposure to arsenic. In addition to the dermal lesions, these individuals all showed vascular lesions (intimal thickening in small and medium sized arteries) (Rosenberg, 1974; Zaldivar, 1974; Zaldivar and Guillier, 1977). The five deaths occurred in children with an age range of 2 to 7 years. The average exposure during the first year of life was 0.13 mg/kg-day. The time-weighted average exposures for the five cases were 0.098, 0.085, 0.081, 0.073, and 0.053 mg/kg-day (Zaldivar and Guillier, 1977).

Follow-up investigations were conducted in Antofagasta, Chile, after a water treatment plant was completed in 1970. Zaldivar (1974) reported that the average arsenic concentration of the drinking water after treatment was reduced from 0.6 to 0.08 mg/L. There was a significant decrease (16 fold) in the average incidence rate of skin lesions in the general prevalence in 1971 as compared to 1968-1969 (Zaldivar, 1974). These data imply that the skin lesions are reversible when exposure is greatly reduced. In addition, Borgono et al. (1977) reported that there were no cases of skin lesions found in 306 children examined who were less than 6 years of age. These children only drank water after the treatment plant was installed. These data imply that a 7.5 fold reduction in the arsenic concentration in drinking water (i.e., 0.6 to 0.08 mg/L) eliminates the risk of acquiring arsenic induced skin lesions in this population.

Hopenhayn-Rich et al. (2000) conducted a retrospective study of late fetal, neonatal, and postnatal mortality in Antofagasta for the years 1950 to 1996. The interpretation of the data is made difficult by the general decline in infant mortality in the region due to changing socioeconomic conditions and improved prenatal health care. Despite this complication, the report documents an elevation in late fetal, neonatal, and postnatal mortality compared to the comparison group in Valparaiso during the period when the drinking water in Antofagasta was contaminated with arsenic (1958 to 1970). There was a decline in late fetal, neonatal, and postnatal mortality after installation of a water treatment plant. After installation of the plant, the mortality rates in Antofagasta were indistinguishable from those in Valparaiso. These data strongly imply that infant mortality potentially associated with exposure to inorganic arsenic does
not occur at an exposure lower than that required to cause the skin lesions characteristic of arsenic toxicity.

Several papers described the investigation of arsenic poisoning from drinking water in West Bengal (Chakraborty and Saha, 1987; Mazumder et al., 1988; Mazumder et al., 1998). The exposure to high concentrations in the drinking water apparently started in the late 1960s. Chakraborty and Saha (1987) reported skin lesions typical of exposure to arsenic in 25% (197/784) of the individuals examined. Cases included children and adults. The average arsenic concentration of the well water for affected individuals was 0.64 mg/L (range 0.2 to 2.0) and for unaffected individuals was 0.2 mg/L (range 0 to 0.74 mg/L). The duration of exposure was reported as 1 to 11 years. The average consumption of drinking water and body weight were not reported, precluding estimating the exposure. These data imply, however, that a three fold reduction in the average arsenic concentration in well water is sufficient to put the exposure into a no effect range.

Mazumder et al. (1998) describe a cross-sectional survey conducted between April 1995 and March 1996 in West Bengal to investigate the prevalence of skin lesions (hyperkeratosis and hyperpigmentation) with increasing exposure to arsenic. In all, 7683 participants were examined and the concentration of arsenic in their drinking water was measured. The average duration of exposure was not reported but could have been up to 25 years (1970 to 1995). The age-adjusted prevalence of hyperkeratosis was strongly related to the concentration of arsenic in drinking water. In the lowest exposure category (<50 · g/L), the prevalence was zero in females and 0.2 per 100 for males; in the highest exposure category (>800 · g/L), the prevalence was 8.3 per 100 for females and 10.7 per 100

for males. The prevalence of hyperpigmentation also showed a strong exposure-response relationship.

The authors report the age-adjusted prevalence of skin lesions in children less than 9 years old (n=1149, male and female combined) as a function of exposure to arsenic in drinking water (mg/L). The average age of the group was not reported. These children are assumed to have been exposed since birth. Among these 1149 children, there were 4 cases of keratosis and 21 cases of hyperpigmentation. The prevalence of skin lesions was also examined by daily exposure per kg body weight and reported by tertile. The exposure was 0 to 0.0032 mg/kg-day, 0.0032 to 0.0149 mg/kg-day, and 0.0149 to 0.0739 mg/kg-day, in the first, second, and third tertile, respectively. EPA calculated the number of individuals in each tertile from the absolute number of cases and the % prevalence reported by the authors. In children less than 9 years old (male and female combined), the prevalence of keratosis was 0/66, 0/66, and 2/66 and the prevalence of hyperpigmentation was 0/66, 1/66, and 5/66 in the first second, and third tertile, respectively. The result was statistically significant ($p<0.05$, one tailed test) only for hyperpigmentation in the third tertile. These data demonstrate a no effect level of 0.0149 mg/kg-day for skin lesions in children. Because the authors included individuals with an exposure of 0.0149 to 0.0739 in one group, a threshold for dermal lesions in this exposure range cannot be determined.

The authors also report standardized morbidity ratios (SMR) for skin lesions for all ages combined as a function of total arsenic exposure for subjects below 80% of the standard body weight corrected for sex, age, and height using those above the 80% value for body weight as the referent population. The purpose of this analysis was to determine if there was an association between nutritional status and the effect of arsenic, using low body weight as a surrogate for impaired nutritional status. The average duration of exposure was not reported; the maximum duration of exposure was 25 years. Individuals (males and females combined) exposed to arsenic were stratified by tertile. Statistical analyses were conducted by comparing the "under nourished" group with the "sufficiently nourished" group in the respective tertile. There was no statistically significant difference in SMR for hyperpigmentation for any tertile. For hyperkeratosis there was no statistically significant difference between the two lower tertiles, but there was a statistically significant difference in the third tertile ($n = 1498$; SMR = 1.7; 95% Confidence Interval = 1.0 - 2.6; $p<0.05$). The exposure in the third tertile was 0.0149 to 0.0739 mg/kg-day. These data show a no effect level for arsenic of 0.0149 mg/kg-day in under nourished individuals, a potentially sensitive sub-population, when the exposure could have been 25 years.

Results from subchronic studies that provide sufficient information to estimate exposure are summarized in the table below. These data support the conclusion that exposure to arsenic at 0.05 - 0.06 mg/kg-day for greater than four months will cause mild skin lesions. There is also some evidence of mild neurological effects if exposure continues for many years.

Exposure quantified	Duration	Effects observed	Cases	Exposure (mg/kg-day)	Reference
Drinking water	4 months	gastrointestinal, neurological, skin	1	>0.05	Wagner, 1979
Fowlers solution	15 months	skin, gastrointestinal	1	0.12	Wade and Fraser, 1953
Fowlers solution	28 months	skin, neurological	1	0.05 - 0.10	Silver and Wainman, 1952
Fowlers solution	3 years	skin, portal hypertension	1	0.08	Morris et al., 1974
Drinking water	6 months - 12 years	skin	150/336	0.05	Huang et al., 1985
Drinking water and food	2-7 years	death	5	0.053 - 0.098	Zaldivar, 1974; Zaldivar and Guillier 1977; Rosenberg, 1974
Drinking water	6 years	skin, neurological	1	>0.09	Foy et al., 1992
Drinking water and food	10 years	skin	3250/27,088 37/300	0.06	Borgono and Greiber, 1972; Zaldivar and Ghia, 1980a, b
Drinking water	0 - 9 years	hyperpigmentation	5/66	0.0149-0.0739	Mazumder et al., 1998

Other studies with a primary focus on lifetime exposure provide some relevant information on shorter-term exposure and a likely no effect level for skin lesions in children. Cebrián et al. (1983) compared the prevalence of signs and symptoms of arsenic poisoning in two rural populations. The arsenic concentration in the drinking water of the exposed populations was 0.41 mg/L. The prevalence of skin lesions in this populations was 21.6% (64/296). The arsenic concentration in the drinking water of the control population was 0.005 mg/L. The prevalence of skin lesions in this population was 2.2% (7/318). The author stated that the daily water intake was 2.5 L for women and 3.5 L for men. The average body weight was not reported. EPA assumed a body weight of 55 kg and reported the estimated exposure to arsenic in the exposed groups as 0.022 mg/kg-day (EPA, 2002). Assuming that

children drink approximately 1.9 times more water in relation to their body weight than adults (EPA, 1997, Table 3-30), the estimated exposure for children would be 0.04 mg/kg-day. Among children 0-9 years of age (assumed to have been exposed since birth), there were no cases of hyperpigmentation or hyperkeratosis and two cases of hypopigmentation reported in the exposed group. The total number of children in this age group was not stated. The shortest time of exposure until a skin lesion appeared was 8 years for hypopigmentation and 12 years for hyperpigmentation and palmoplantar keratosis. These data suggest that an exposure duration of seven years at an exposure of 0.04 mg/kg-day is a no-observed adverse effect level for skin lesions in children. However, Cebrián et al. (1983) is not used as a primary report because the number of individuals exposed was not stated and EPA cannot judge the power of the study.

The data in Tseng (1977) and Tseng et al. (1968) were used to establish EPA's chronic reference dose. The lowest-observed-effect level for skin lesions (hyperpigmentation and hyperkeratosis) was established at 0.014 mg/kg-day (EPA, 2002). Although the authors did not present data on the prevalence in specific age groups or at specific concentrations of arsenic in drinking water, the data show a very strong increase in prevalence of skin lesions (hyperpigmentation, keratosis) and vascular lesions (Blackfoot Disease) with increasing duration of exposure. See Tseng et al. (1968) Figures 5, 6 and 7, and Tseng (1977) Figure 6 and Table 2. This study examined more than 14,000 children less than 10 years of age who had consumed arsenic containing water from birth and the data suggest that there was no response or a very minimal response in this age group. The concentration of arsenic in the water ranged from 0.01 - 1.82 mg/L, with the majority around 0.4 - 0.6 mg/L. EPA estimated the average exposure for a 55 kg adult as 0.014 mg/kg-day (EPA, 2002). Assuming that children drink approximately 1.9 times more water in relation to their body weight than adults (EPA, 1997, Table 3-30), the estimated exposure for children would be 0.03 mg/kg-day. These data suggest that an exposure duration of ten years or less at an exposure of 0.03 mg/kg-day is a no-observed adverse effect level for skin lesions in children. However, Tseng (1977) and Tseng et al. (1968) are not used as primary reports because the number of individuals exposed at each arsenic level was not stated and EPA cannot judge the power of the study.

Conclusions on Exposure-Response

Taken together the primary data reported in the above tables establish that exposure to arsenic at 0.05 - 0.06 mg/kg-day from drinking water or other sources with readily bioavailable arsenic for one day to ten years will cause adverse effects. The effect level is approximately the same from acute and subchronic exposure. Skin lesions (hyperpigmentation and hyperkeratosis) are the most consistent finding at this exposure, with some suggestion of gastrointestinal and neurological effects in several cases. The reports included a large number of people (more than 42,000) in diverse populations and in different parts of the world. In addition, the reports included young children and undernourished individuals, which are potentially sensitive subgroups. In most of the large cross-sectional epidemiological studies, where exposure was for ten years or less at 0.05 - 0.06 mg/kg-day, the prevalence of skin lesions was approximately
12-25% of the population examined and of minimal severity. The data in one report establish a no-observed adverse effect level in children and potentially undernourished individuals when exposure was

0.0032 - 0.0149 mg/kg-day (Mazumder et al., 1998). Data from other reports (Cebrián et al., 1983; Tseng, 1977; Tseng et al., 1968) imply that a no-observed adverse effect level might be as high as 0.03 - 0.04 mg/kg-day when exposure is seven to ten years or less.

Although there are no reports where exposure was stopped and the population followed for years to determine whether latent effects occur, circumstantial evidence from the reports in Chile (Zaldivar, 1974) and China (Huang et al., 1985) suggest that the skin lesions regress or disappear when exposure stops.

Derivation of Acute and Subchronic Oral Reference Dose

When human data are available to derive an oral reference dose, EPA may apply uncertainty factors to account for extrapolation from a LOAEL to a NOAEL, for potentially sensitive individuals, and for an incomplete data base. The magnitude of each uncertainty factor is typically 1, 3, or 10 depending on the data and professional judgment.

As discussed above, there are many human studies demonstrating a LOAEL at 0.05 - 0.06 mg/kg-day from acute and subchronic exposure and less robust evidence showing a NOAEL in the 0.015 - 0.04 mg/kg-day range from subchronic exposure. The LOAEL was chosen as the point of departure for deriving an RfD because there is consistent evidence across numerous studies that adverse effects occur at or slightly above this exposure level, while the data to establish a definitive NOAEL is less conclusive.

With regard to the LOAEL to NOAEL extrapolation, an uncertainty factor of 3 is often used when the data demonstrate a low incidence of response and it is likely that a NOAEL is only slightly below the LOAEL, or the effect observed is of minimal severity. An uncertainty factor of 10 is usually applied in cases where there is inadequate information to characterize the exposure-response relationship below the LOAEL, where the critical effect cannot be judged as minimal in severity, or where a somewhat higher exposure is likely to produce a more severe effect.

In the case of arsenic, an uncertainty factor of 3 is used for extrapolation from a LOAEL to a NOAEL. Hyperpigmentation and hyperkeratosis are the primary adverse effects observed at 0.05 - 0.06 mg/kg-day. These dermal effects were generally of minimal severity and the prevalence is relatively low at this exposure, but it should be noted that the effects may not be completely reversible and there is no definitive data establishing that the skin lesions do not appear after exposure ceases. More severe effects (abnormalities in electrocardiogram and peripheral neuropathy) have been documented at exposures of 0.11 - 12 mg/kg-day (Barbey et al., 2001; Soignet et al., 2001; Wang, 2001). Mizuta et al. (1956) also reported electrocardiogram abnormalities and mild peripheral neuropathy at a dose of 0.05 mg/kg-day, but there is significant uncertainty regarding this exposure level. The mild nature of the adverse effect (skin lesions) and the evidence showing that a NOAEL is likely at an exposure level only slightly below the LOAEL of 0.05 - 0.06 mg/kg-day justifies the application of an uncertainty factor of 3 to the LOAEL, rather than the default uncertainty factor of 10 (see the discussion above of Zaldivar (1974); Chakraborty and Saha (1987); Mazumder et al. (1998); Cebrián et al. (1983); Tseng (1977) and Tseng et al. (1968)).

An uncertainty factor for sensitive individuals (i.e., intraspecies or human variability) is not used

because the epidemiological studies and case reports included a large number of individuals, diverse populations from different parts of the world, various life stages including children, and undernourished individuals. These populations likely included potentially sensitive subgroups.

An uncertainty factor of 3 is used for data base limitations. The available data primarily address dermal effects and the exposure-response relationship has not been adequately characterized for other adverse effects (e.g., cardiovascular and neurological) in the most susceptible population. While the data suggest that responses to arsenic exposure in children and adults are qualitatively and quantitatively similar, information on neurological effects in children at exposures at or near the LOAEL is lacking, including possible neurodevelopmental effects. There are also no definitive data on possible latent effects in children. Last of all, toxicokinetics information in children is limited and it is unclear whether age-dependent differences in toxicokinetics may affect toxicity in children.

The acute and subchronic reference dose for inorganic arsenic is 0.005 mg/kg-day, which is derived from the LOAEL of 0.05 mg/kg-day divided by a total uncertainty factor of 10. The total uncertainty factor is based on a factor of 3 to account for the LOAEL to NOAEL extrapolation and a factor of 3 for database uncertainties. The acute reference dose applies to an exposure of 1 to 14 days, while the subchronic reference dose applies to an exposure of 14 days to seven years. For an exposure longer than seven years, EPA's chronic RfD of 0.0003 mg/kg-day for inorganic arsenic is the appropriate value.

The acute and subchronic reference dose applies to readily soluble forms of arsenic and is intended to include total oral exposure to inorganic arsenic, including drinking water, food, and soil. Inorganic arsenic in drinking water is considered totally bioavailable. The average U.S. diet for an adult contains 0.01 to 0.015 mg/day of inorganic arsenic that is also considered totally bioavailable. Any exposure assessment for soil should account for the relative bioavailability of arsenic using site-specific data. In the exposure assessment, it is also important to consider if there are individuals who would have a higher than normal exposure to inorganic arsenic (i.e., children exhibiting soil-pica behavior, individuals with a greater than average exposure to inorganic arsenic in food, or adults with exposure to inorganic arsenic in the work place).

ATSDR Oral Minimum Risk Level (MRL)

ATSDR established a Provisional Acute Oral MRL that applies to an exposure of 1 to 14 days based on the effect level of 0.05 mg/kg-day from Mizuta et al. (1956). Using an uncertainty factor of 10 for the extrapolation to a no-observed adverse effect level, the Provisional Acute Oral MRL is 0.005 mg/kg-day. ATSDR did not establish an Intermediate Oral MRL that apples to an exposure of >14 days to 1 year. ATSDR established a Chronic Oral MRL that applies to an exposure of 1 year or more. ATSDR's Chronic Oral MRL is 0.0003 mg/kg-day, using the same information and uncertainty factor used by EPA to establish its Chronic Reference Dose (RfD). Regarding the studies used to establish the chronic Oral MRL, ATSDR states "collectively, these studies indicate that the threshold dose for hyperpigmentation and hyperkeratosis is approximately 0.01 mg As/kg-day" for chronic exposure.

Regarding the definition, interpretation, and use of MRL values in risk assessment and risk

management, ATSDR uses the substance specific MRLs as screening levels to identify contaminants for further evaluation in its public health assessments and to identify potential health effects that may by of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels. They are health guidance values below which non-cancer adverse effects are unlikely and are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. Exposure to a level above the MRL does not mean that adverse health effects will occur. MRLs are intended only to serve as a screening tool to help public health professionals decide when a more detailed toxicological evaluation is necessary. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects in an exposed population. When using MRLs, public health officials should realize that they do not cover exposure to multiple chemicals nor do they cover cancer effects.

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